TRANSACTIONS

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and
OTOLARYNGOLOGY

MAY - JUNE
1955

Volume 59 Number 3

ANNUAL MEETING, OCTOBER 9-14, 1955, CHICAGO

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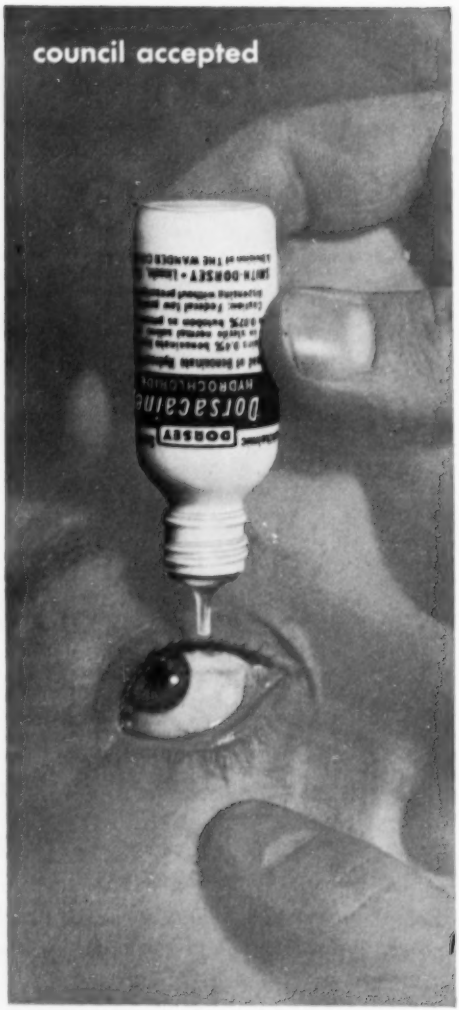

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*Schlegel, H. E., Jr., and Swan, K. C.: A. M. A. Arch. Ophth. *51*:668 (May) 1954.

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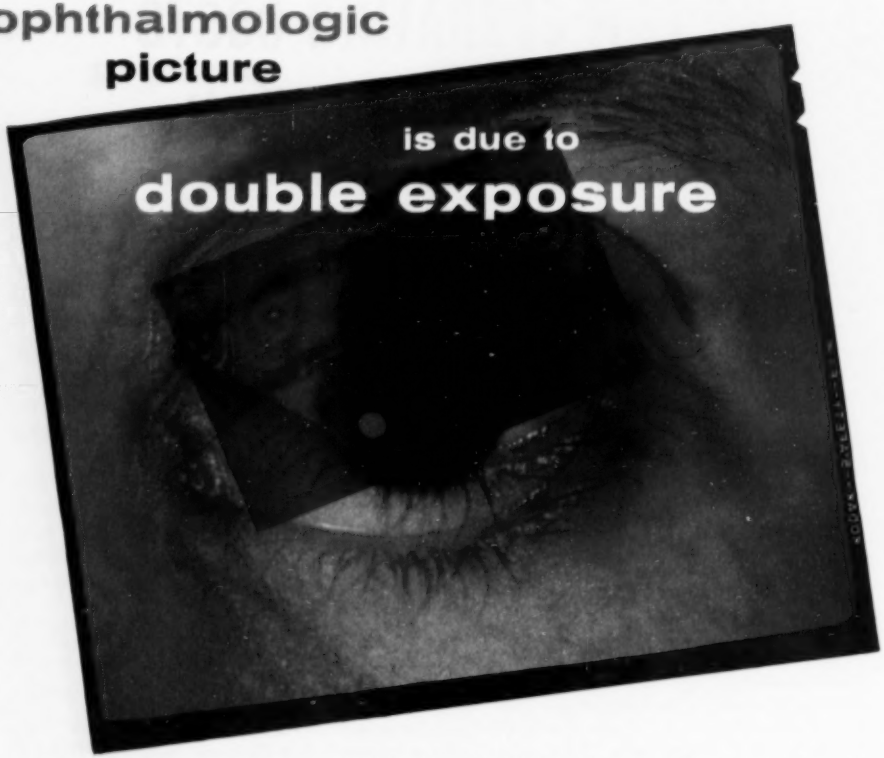

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1. Rukes, J. M., et al.: Metabolism 3:481, 1954. 2. Cannon, E. J., and Leopold, I. H.: A.M.A. Arch. Ophth. 47:426, 1952.

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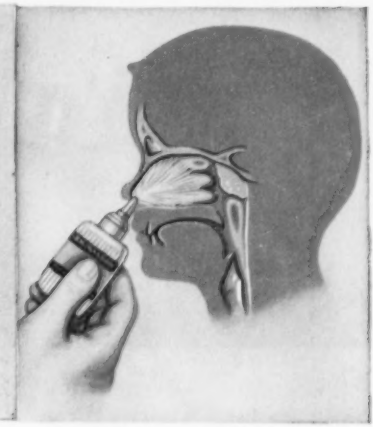

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7. Kugelmass, I. Newton: Handbook of the Common Acute Infectious Diseases, 1949.

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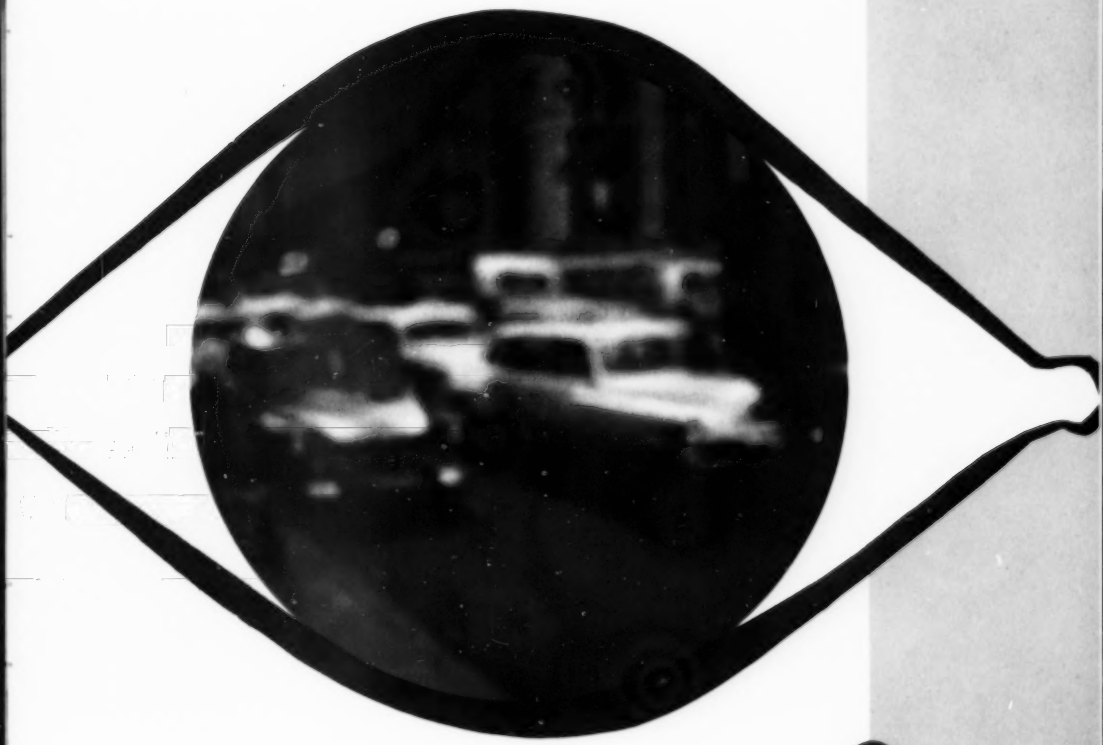

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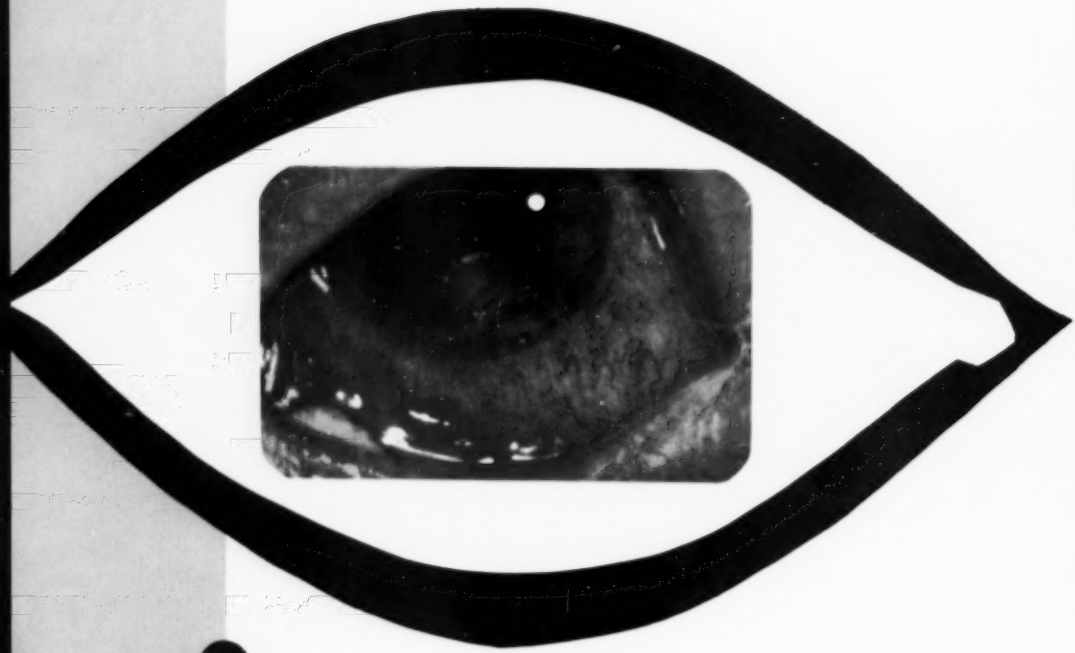

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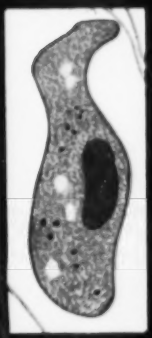

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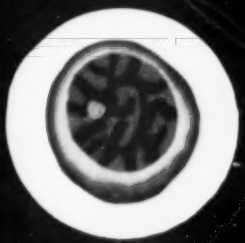

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(1) Leech, C. A., Jr.: Clin. Med. 2:255, 1955. (2) Lincoln, C. S., Jr.: M. Times 83:178, 1955. (3) Aronoff, S., and Ghaemi, A.: J. M. Soc. New Jersey 50:564, 1953. (4) Fenton, M. M.: Ann. Allergy 11:336, 1953.

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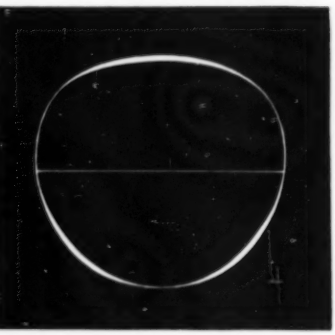

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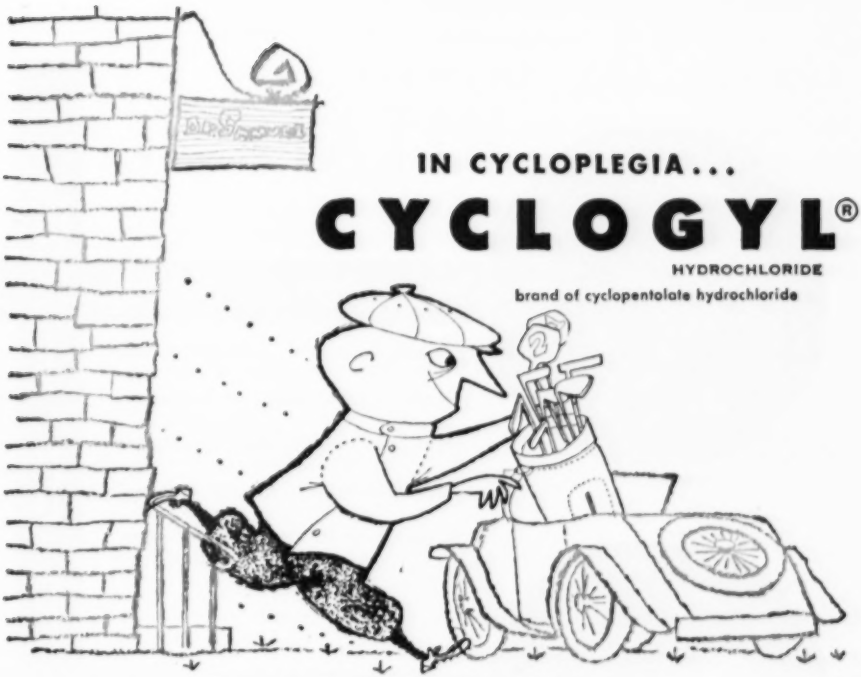

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1. Gordon, D. M., and Ehrenberg, M. H.: Am. J. Ophth. 38:831 (Dec.) 1954.

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Getter, B. C.: A.M.A. Arch. Ophth. 51:467 (Apr.) 1954.

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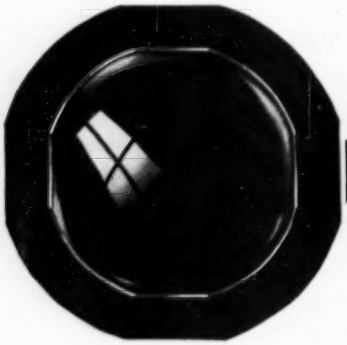

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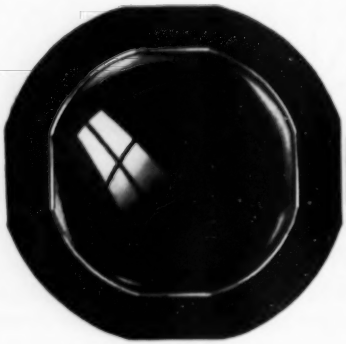

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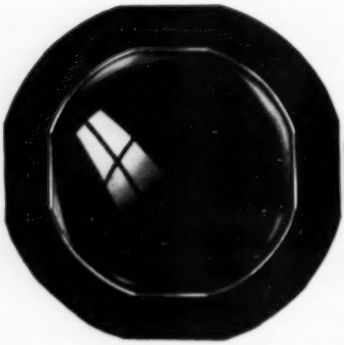

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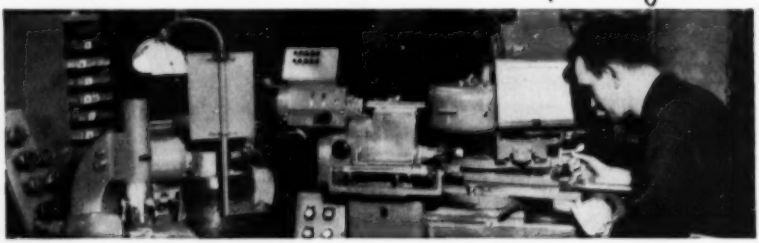

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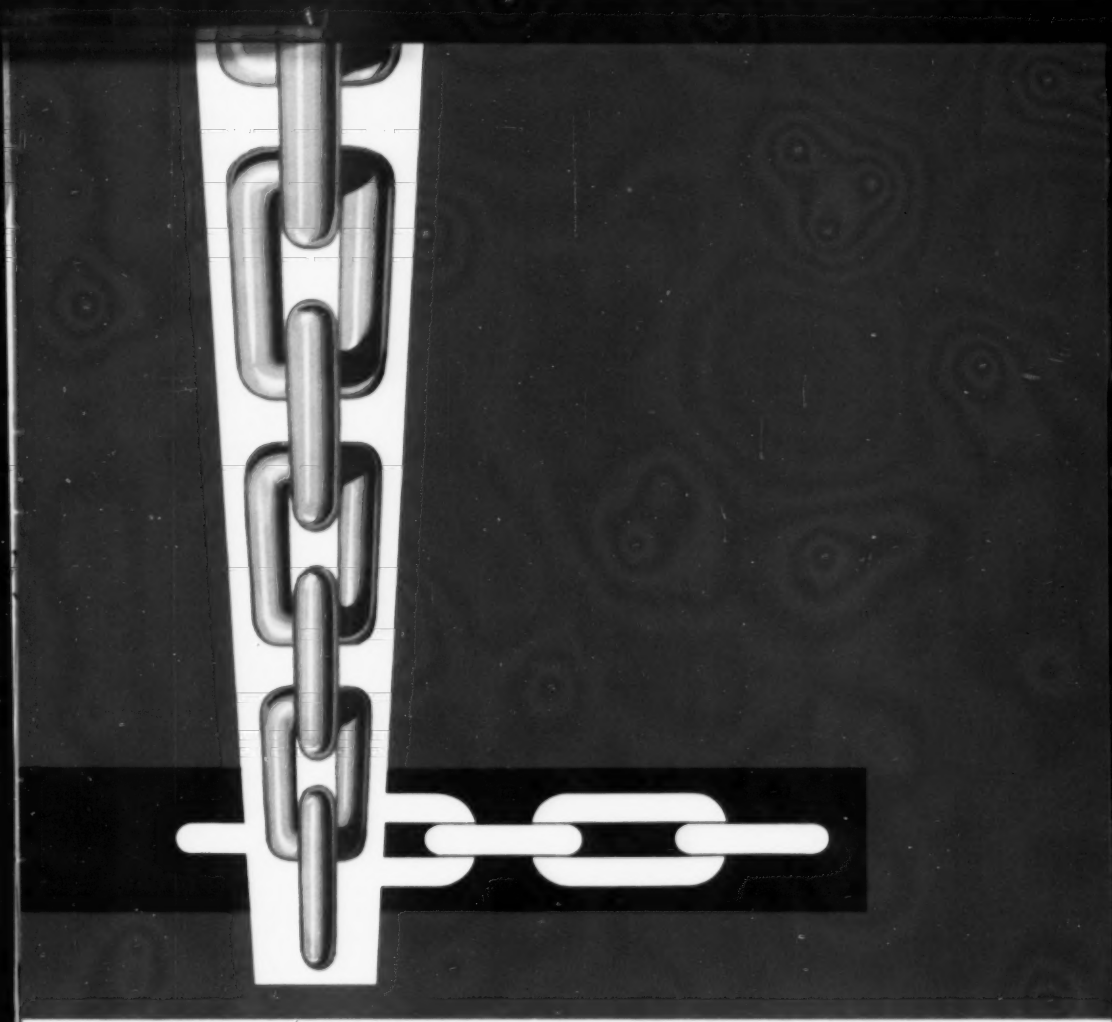

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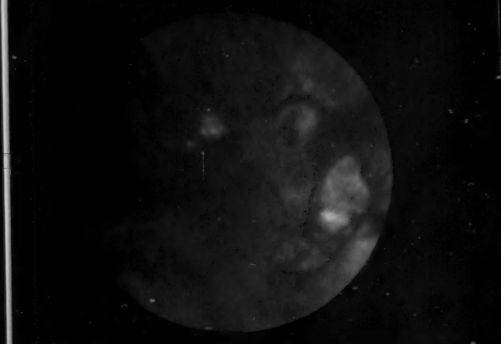
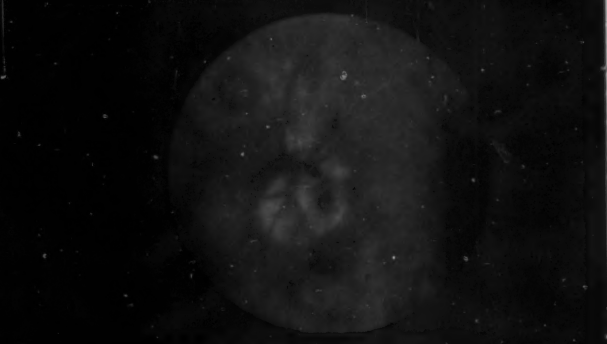


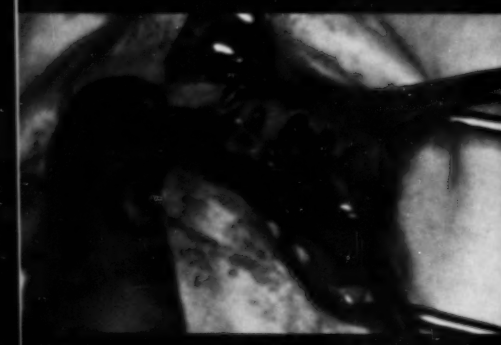
Anticipation of recurrent hemorrhage and visual defect is high in hypertensive patients.



After Adrenosem therapy. Note exudative areas. Maintenance therapy checks further hemorrhage.

Mastoidectomy with a bloody field obscures view, requires frequent suctioning.

After Adrenosem therapy. Note clearer operative field with minimized bleeding.



Multiple extraction without Adrenosem. Note seepage. Frequent suctioning is required.

After Adrenosem therapy. Note clearer field. Minimal bleeding facilitates surgery and promotes healing.

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Adrenosem is specific for conditions characterized by increased capillary permeability allowing oozing and seepage bleeding. To prevent seepage bleeding due to capillary permeability it is necessary to maintain tone of the capillary wall. This is supported by Pappenheimer's[1] work on capillaries. In cases of capillary permeability it is postulated that the connective material between the cells, which form the capillary wall, has lost its tone and allows the erythrocytes to pass through.

To confirm the action of Adrenosem in establishing capillary integrity the cheek pouch of a hamster was irritated with snake venom, which has the property of making the capillaries more permeable.[2] A great many petechiae appeared on the surface and a 500 X magnification showed the capillaries with extravasation of erythrocytes. Another animal was treated with Adrenosem, and 30 minutes later, the same amount of snake venom was used to irritate the cheek pouch. Microphotographs of the same 500 X magnification show the intact capillary walls and the actual blood cells coursing through the capillaries, without signs of escaping erythrocytes.

Fig. 1—*Trauma from snake venom produces hemoconcentration and stasis, indicative of altered vascular permeability.*

Fig. 2—*Trauma does not produce petechiae following Adrenosem injection.*

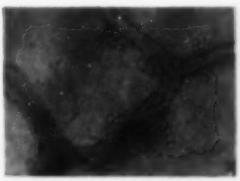

Fig. 1

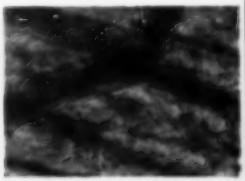

Fig. 2

Action

A systemic hemostat, Adrenosem restores capillary integrity by maintaining tone of intercellular tissue in the capillary wall. Adrenosem is unlike any hemostat available to medicine.[3,4] It does not interfere in any way with the blood clotting mechanism or its component parts. There is no sympathomimetic action and the blood pressure, cardiac rate and volume are not affected. Adrenosem may be administered simultaneously with any type of anesthetic, anticoagulant or vitamin K and heparin. Adrenosem will not interfere with the action of these agents.

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While the hemostasic action of epinephrine and related compounds has been recognized for a long time, certain characteristics limited their usefulness. A product of the metabolism of epinephrine seemed to offer the most promise. Given the name, Adrenochrome, this compound was studied extensively in both European and American centers. The handicap to wide use of Adrenochrome was its instability, so efforts were directed to find a stable modification that would retain the physiological activity. The answer to this proved to be the monosemicarbazone of Adrenochrome. Further research demonstrated that this compound could be made soluble enough to be practicable as an injectable solution, by transforming it to a sodium salicylate complex. This process has made possible Adrenosem Salicylate, a potent therapeutic agent which fills an important need in medicine.

1 *Pappenheimer, John R.: Passage of molecules through capillary walls, Physiol. Rev. 33:387-423, 1953.*

2 *Fulton, M. D., Department of Biology, Boston University: Personal communication, 1954.*

3 *Sherber, Daniel A.: Adrenochrome complex in the control of bleeding, Am. J. Surg. 86:331-335, 1953.*

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5 *Peele, J. G.: Adrenosem in the control of hemorrhage from the nose and throat (in press).*

6 *Bourgoyne, J. R. and Riddle, A. C., School of Dentistry, University of Tennessee: Personal communication, 1954.*

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• preoperatively to control oozing during surgery and provide clearer operative field:
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• postoperatively to prevent hemorrhage and check oozing:
one to two ampuls (5 to 10 mg.) every two hours until there is no indication of undue bleeding. To maintain control, one ampul may be administered every three hours or 1 to 5 mg. orally t.i.d.

• to control active bleeding:
one ampul (5 mg.) every two hours until bleeding is controlled; frequency of dose may then be diminished.

• to control mild, low-grade bleeding:
one ampul (5 mg.) every three or four hours until bleeding is controlled; then 1 to 5 mg. orally four to five times daily until bleeding ceases.

• maintenance dosage to prevent bleeding in conditions where small vessel integrity may be impaired:
1 to 5 mg. orally t.i.d.; if bleeding ensues, dosage may be increased 1 to 5 mg. orally every three or four hours; if bleeding persists, oral dosage should be supplemented with one ampul (5 mg.) daily.

• pediatric dosage:
up to four years of age—1 mg. intramuscularly or orally with same frequency as for adults, until bleeding is controlled. From four to twelve years of age—one-half the adult dosage as indicated.

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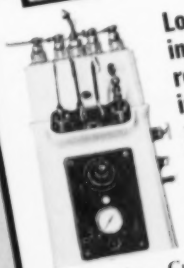

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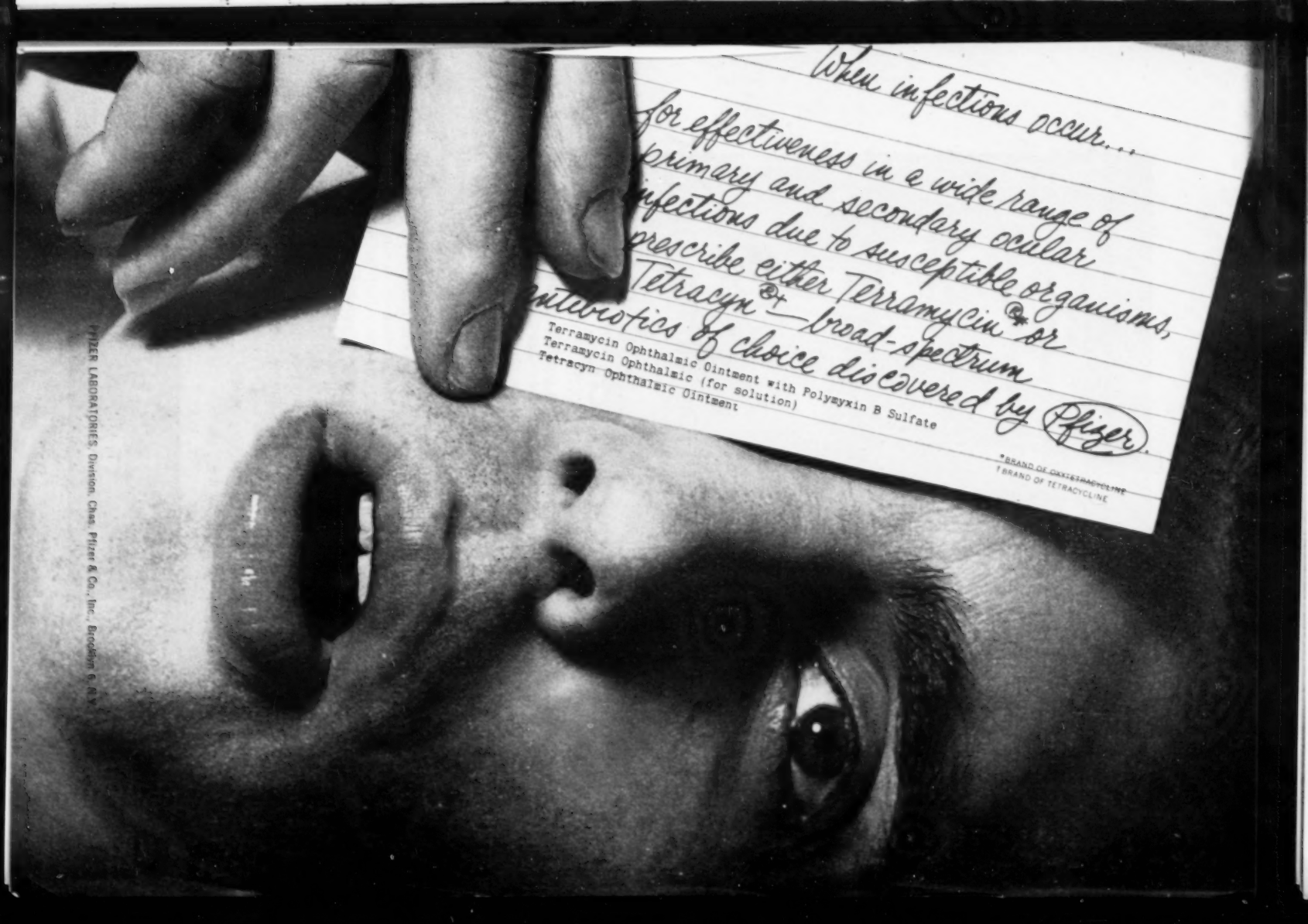
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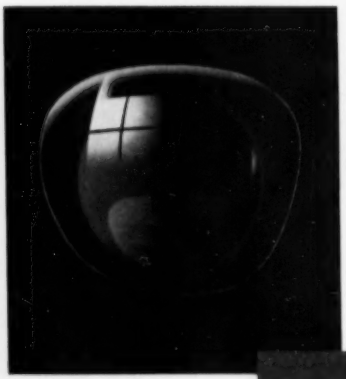

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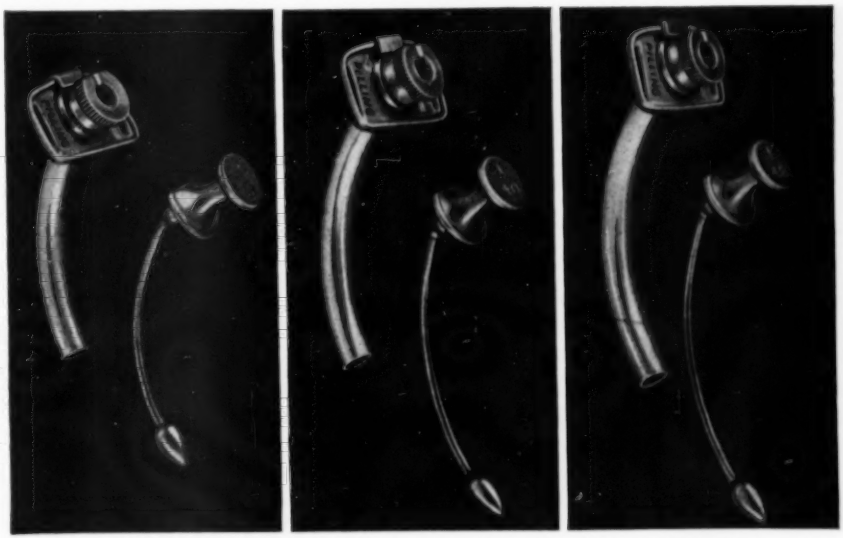

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Jackson Memorial Lecture

PERSISTENT HYPERPLASTIC PRIMARY VITREOUS

ALGERNON B. REESE, M.D.
NEW YORK, NEW YORK

THE Jackson Lecture, by virtue of its symbolic commemoration and the high standard set by the previous lectures, has come justly to its present high estate. I am particularly eager that this lecture should be found worthy of the occasion because it was at this meeting just 50 years ago that Dr. Edward Jackson presided as the first president of this society under its present name. Doctor Jackson was elected a member of the Western Ophthalmic and Oto-Laryngologic Association in 1902 and took a very active part in the scientific program. In 1903, Dr. Casey Wood's report, as chairman of a committee to draw up a new constitution and by-laws, was adopted. This included changing the name of the organization to "The American Academy of Ophthalmology and Otolaryngology." At the first meeting in Denver, Colorado, August 1904, Doctor Jackson was president. Dr. Derrick T. Vail of Cincinnati was secretary. The presidential address was "Education for Ophthalmic Practice." On this occasion, as well as on others, Doctor Jackson championed the appraisal by a suitable board of one's fitness to practice ophthalmology, thus presaging the American Board of Ophthalmology. He also decried the tendency to overspecialization. In this regard it may be that I am an offender today.

From the Institute of Ophthalmology of the Presbyterian Hospital, New York.
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We have employed the term leukokoria (white pupil) for the group of conditions in infants and children frequently referred to as pseudoretinoblastoma. My interests have been in several conditions belonging to this group, and even at the risk of being branded a leukokorologist, the epitome of overspecialization, I wish to discuss still another one of the group — persistent hyperplastic primary vitreous.

Persistent hyperplastic primary vitreous and persistent hyperplastic vitreous (PHV) are the terms used to designate the condition often called "persistent tunica vasculosa lentis" or "persistent posterior fetal fibrovascular sheath of the lens." The tunica vasculosa lentis is actually a very meager layer of small vascular channels surrounding the lens and stemming from three sources: from the hyaloid artery, from the vasa hyaloidea propria (these are derived from the vessels which pass into the peripheral parts of the vitreous and enter the vascular tunic of the lens at its equator), and lastly, from the anterior ciliary vessels which stem from the major arterial circle of the iris. The anterior part of this vascular tunic of the lens is supplied by the ciliary system, while the posterior portion is fed by the hyaloid system. The former (anterior part) normally shows a complete regression at about the eighth month of fetal life, although it is not at all unusual, particularly in premature infants, to see some evidence of this system at birth. The latter (posterior part) usually shows

complete regression at about the seventh fetal month, but remains of this system are also commonly seen at birth, particularly in premature infants. If hyperplasia does not occur, both systems may achieve continued and complete regression after birth. Clinically speaking, the two portions seem to have little or no relationship; persistence and hyperplasia of the one occur independently of the other. There are two aspects of the posterior system — the fibrovascular sheath and the hyaloid artery (figs. 1, 2, 3, 4). When remains of the sheath are present, we commonly see remains, too, of the hyaloid artery or of some feature of the hyaloid system. Also, when the hyaloid artery dominates the picture we sometimes see some degree of the remains of the posterior fibrovascular sheath of the lens. This association of the two portions is not surprising since both are remains of elements of the primary vitreous. One or the other, however, usually predominates.

When retrolental fibroplasia became a major problem about 1942[37-40] it was thought to be related to PHV. Further experience proved, though, that the two conditions are separate entities.[28]

MATERIAL

Because of an interest in both retinoblastoma and retrolental fibroplasia, I have had the opportunity to see a relatively large number of allied conditions. Therefore, my series of clinical cases of persistent hyperplastic vitreous in various stages numbers 59, and in many instances I have been able to follow the lesion from its detection to the ultimate fate of the eye. I am prompted to report my experience with these cases because I have been struck (1) by the wide clinical variations which they manifest and (2) by the fact that this bizarre clinical picture is frequently not appreciated. Furthermore, the eyes harboring these lesions usually are ill-fated. As a matter of fact, I have not been able to discover a single recognizable case in an adult. It is true that mild lesions of this type may run an uncomplicated course and be seen occasionally in adult eyes. It has been challenging to undertake measures to prevent the complications which lead ultimately to the loss of such eyes. It is possible that I have been successful in some degree in this aspect of my endeavor.

As eyes with PHV either are often removed with a diagnosis of retinoblastoma or develop complications which lead to enucleation, the pathological material for study is rather rich. It has been interesting, therefore, to try to correlate the clinical observations with the pathological material. I have had the opportunity to study microscopic sections of 47 eyes* with PHV of varying degrees of severity at various stages in the course of the disease. Twenty-two eyes were available from the pathology collection of the Institute of Ophthalmology and 25 eyes have been studied through the courtesy of the Armed Forces Institute of Pathology.

PATHOLOGY

The fibrous tissue back of the lens varies a great deal in extent and thickness (figs. 4, 5, 6, 7, 8). It not only may cover the entire posterior surface of the lens but may even extend from one ciliary region to the other; or it may occupy only a portion of the lens surface on the nasal side (fig. 6). The thickness may vary from that of a thin membrane to a thickness as great as, or greater than, that of the lens itself. The central portion is usually thicker than the peripheral. There are various degrees of vascularization, and sometimes in this

*Also, I have a dog's eye with typical PHV. The globe was enucleated because retinoblastoma was suspected.

tissue there is evidence of recent or old hemorrhage and blood pigment. There are some (Heine,[12] Hamma,[13] Lane[20]) who believe that hemorrhage plays an important role in producing the fibrovascular sheath. Fat, smooth muscle, calcium deposits, cartilage, eosin-staining hyaloid material, and islands of undifferentiated neuroepithelium may be present. The fibrous tissue seems to be composed of connective tissue (van Gieson and Mallory stains) although it is possible that there are sometimes glial components (von Hippel[16]). The lens zonules may be inserted into the periphery of the fibrovascular sheath (Collins,[2] von Hippel[16]). The pathway of the venous return from the fibrovascular sheath is highly speculative (von Hippel[16]). Very rarely, fibrous filaments extend from the posterior surface of the retrolental tissue towards the retina and may even pull the retina away from the choroid. These extensions are evidently remains of the vasa hyaloidea propria. It is extremely uncommon for the retina to be detached and lie in folds around the fibrovascular sheath; this occurrence seems to be due to spontaneous hemorrhage and organization (fig. 25).

The hyaloid artery can usually be seen where it enters the posterior surface of the tissue in the central part on the nasal side. This artery may be patent or atresic. It usually has around it an easily detectable layer of smooth muscle which extends into and along the fibrovascular sheath.

The lens is small and sometimes shows a posterior concavity (Pollock[27]), giving it a kidney shape. One of the most striking findings is a central opening in the posterior capsule (Parsons,[26] Collins,[3] Pollock,[27] Leech,[21] Seefelder,[33] Sanders,[32] von Hippel,[16] Marshall[23]). This break in the capsule can be seen in almost every eye harboring PHV (figs. 5, 7). Pollock[27] felt that the capsular rent occurs about three months after birth. Collins[1] believed that it occurs early in fetal life and may be the cause of the persistent and hyperplastic fibrovascular sheath and hyaloid artery. My impression is that it usually occurs after birth, but this opinion is based on clinical observations of cases in which the lens is at first clear but later develops cataractous changes. The lens epithelium, the fibrous tissue, and the blood vessels tend to proliferate into the lens through the opening in the posterior capsule. Hemorrhage sometimes occurs inside the lens (Parsons,[25] Collins,[3] Lane[20]). There usually is a wrinkling of the posterior capsule. This wrinkling is apparently due to contracture of the fibrous tissue along its posterior surface. It may be, therefore, that the contracting fibrous tissue is responsible for the opening which occurs in the capsule. Another frequently observed feature is the extension of the lens epithelium under the capsule posteriorly (Gifford and Latta,[9] Pollock,[27] Sanders,[32] Dötsch,[6] Ginsberg[10]). Not only is the lens smaller in diameter and thinner than normal, but also there is a gradual absorption of the lens substance due to the opening in the posterior capsule. Ultimately the lens may be totally absorbed (figs. 17, 26) (Findlay,[8] von Grolman,[11] Czermak[5]) with only the capsule remaining in a bed of fibrous tissue, or the lens may shrink to a small globule of old calcified cortex surrounded by the capsule.

Elongated ciliary processes are a constant finding. They usually extend as far as the equator of the lens and they are frequently incorporated in the periphery of the fibrovascular tissue. They are hyperemic and their stroma is sometimes replaced by dilated vascular channels. Ida Mann, in discussing Hudson's paper,[19] felt that long, drawn-out ciliary processes are often seen in maldeveloped

eyes, particularly in conjunction with congenitally malformed lenses. She thought they are due to adhesion of the processes to the vascular sheath (normal to about the fifth month). If this adhesion persists, the processes are drawn out in the subsequent growth of the eye.

There may be evidence of chronic inflammation in these eyes (Findlay,[8] Leech,[21] Pollock[27]), but it is noted early in the eyes in which lens absorption is taking place. Leech (and also Pollock) felt that such inflammation indicated that a fetal iritis had caused the PHV. I feel sure, however, that this inflammatory reaction occurs in response to the presence of the lens matter.

Characteristically, the retina in these eyes is in place and contains all of its nuclear and fibrous elements as well as a well formed fovea. There is no reason, therefore, to believe, judging from the appearance of the retina, that it would not be capable of good function. A common feature is an extension of the retina peripherally towards the flat portion of the ciliary body and even to the ciliary processes. Sometimes the retina extends, or is pulled, into the periphery of the fibrous tissue back of the lens, usually without promoting an actual detachment of the retina.

The filtration angle usually shows some degree of malformation (Terry,[41] Paganelli[24]). This is in the nature of an embryonic type of angle wherein the iris processes form a bridge between the cornea and iris. Sometimes the trabecular spaces and Schlemm's canal do not appear patent.

Hemorrhage may be a feature in the more advanced cases and is seen not only in the lens and in the tissue back of the lens but also, in various stages of organization, in the vitreous. This may cause the retina to be detached and drawn up into the organizing and contracting tissue (fig. 25).

Clinical observations and a study of available pathologic material indicate that eyes with PHV are lost in two ways. One way is through swelling of the lens due to an opening in the posterior capsule, and the other is from spontaneous hemorrhage. These two factors will be amplified later.

CLINICAL COURSE

In clinical appearance this lesion assumes the most protean forms. This is due in part to varying severity, but it is also due to the facts (1) that the process is never static but always changing and (2) that the natural course leads to sequelae and complications. The lesion occurs in full-term infants of normal birthweight with no unusual features during the prenatal and postnatal course.

The presence of the lesion is usually noted by the physician or the parents immediately after birth or within a few weeks thereafter. In 90 per cent of the cases the lesion is unilateral with a perfectly normal fellow eye, or merely a Mittendorf dot or some such slight manifestation in the fellow eye. The affected eye has a shallow anterior chamber and is almost invariably smaller than its fellow. (I have seen two exceptions to this.) The degree of microphthalmos may be so slight, however, that it is not appreciated unless looked for or unless the cornea is actually measured. Some of the patients have nystagmus (Marshall,[23] Gifford and Latta[9]) and the affected eye may show an esotropia or, rarely, an exotropia.

The tissue back of the lens (figs. 9, 10, 20), usually densest in the center and thinning towards the periphery, has a pinkish white color and varies in size from just a small plaque (located nasal-

ly to the center of the lens) to an area completely covering the posterior surface of the lens. There is always some vascularity of this tissue, and vessels frequently radiate from the center. When the pupil is dilated, the ciliary processes can be seen extending towards, into, or anterior to, the periphery of the opaque tissue. These long ciliary processes are a characteristic feature of the disease and often serve as a differentiating point in the diagnosis. The fibrous tissue undergoes gradual contracture, which draws the processes centrally and makes them clearly visible (figs. 16, 20). Hemorrhage, which is sometimes seen in or around the tissue (figs. 13, 18, 20), seems to appear several months (most often around four months) after birth.

The lens characteristically is smaller in diameter (Findlay,[8] Hudson[19]) than would be normally expected even in a somewhat microphthalmic globe. For this reason, the equator of the lens can frequently be seen easily, and occasionally it is inside of, and concentric to, the pupillary margin when the pupil is dilated (figs. 10, 12, 20). Sometimes the lens border protrudes through the dilated pupil, particularly on the nasal side (fig. 12). In such instances the iris, when not dilated, protrudes cone-shaped. The lens is at first perfectly clear and later becomes cataractous (figs. 9, 10, 11, 12). These cataractous changes make their primary appearance at the posterior pole (fig. 11) where the opacity mushrooms forward into the still transparent portion of the lens. This opacity gradually increases until the entire lens is opaque. The development of the cataractous change is accompanied by a swelling of the lens, which frequently comes forward, together with the iris, and touches the cornea (figs. 4, 7, 12, 21, 22, 23, 24). This development is accompanied by secondary corneal changes in the form of edema and, later, of scarring (figs. 13, 14, 18). Such complications are usually followed by glaucoma. If, on the other hand, the swelling of the lens is not sufficient to cause its apposition to the corneal surface and to provoke secondary glaucoma, the lens may undergo absorption and leave only the capsular remains over the surface of the retrolental tissue (figs. 15, 16, 17, 26). Or, the lens may undergo partial absorption and be seen as a contracted globular mass, usually calcified, lying in the central part of the concave tissue (figs. 18, 19). Occasionally a posterior lenticonus can be appreciated before the lens becomes opaque (Steffen,[35] Seefelder,[33] Cordes,[4] Collins[1]). Hemorrhage may be seen rarely inside the lens (from the fibrovascular sheath which proliferates into the lens substance through the opening in the posterior capsule) associated with or without siderin and crystals. These by-products of hemorrhage may be seen to emerge into the anterior chamber when the lens capsule is opened at the time of a needling operation. The crystals may be seen over the iris and in the pupillary area for a long time thereafter.

The iris usually shows abnormally large and numerous blood vessels, most of which course radially (Hudson,[19] Leech[21]). Sometimes it shows one or more large anomalous vessels which may extend onto the surface of the lens or around the pupil to be lost in the posterior chamber, or they may be seen to anastomose with the blood vessels in the retrolental tissue. Ida Mann, in discussing Hudson's paper,[19] stated that the iris vascularity in these cases resembled that seen in the fetus from the seventh to the eighth month, when all vessels are visible. The iris often dilates rather poorly with mydriatics (Lent and Lyon[22]). In the more advanced cases there may be ectropion and atrophy of

the iris. Remains of the fetal pupillary membrane have not been noted as a feature of the disease.

Glaucoma is a common complication and, as stated, it seems to be caused usually by the swelling of the lens. It is doubtful that the cause is malformation of the filtration angle because glaucoma is not encountered in the early stages, but only late in the course when the anterior chamber becomes more shallow or is even obliterated by a swelling of the lens. A second cause of the glaucoma may be spontaneous hemorrhage in the vitreous or in the perilenticular area. Iris bombé may occur from posterior synechias and promote glaucoma. If the glaucoma continues, buphthalmos ensues. These buphthalmic eyes frequently exhibit an opaque cornea due to the apposition of the lens and iris to its posterior surface. The globe may then proceed to atrophy, possibly with band keratopathy (figs. 10, 14).

NATURAL HISTORY OF THE DISEASE

In lesser degrees of the lesion, i.e., when only a small patch of tissue lies along the posterior surface of the lens, the progressive changes noted are merely those secondary to contracture of the retrolental tissue. They manifest themselves by a gradual pulling of the ciliary processes further and further centrally and by a shrinkage of the lens. Therefore, some cases in which there have been minimal changes and which have not developed serious complications may be seen years later. I have followed a case of this kind from the birth of the patient, 14 years ago, until the present. This is the only case I have seen which has not developed complications leading to the loss of the eye except the treated cases and the three cases discussed in Group II, on page 281. However, these lesser manifestations of the disease are so rare that we can assume that the great majority of eyes affected with PHV are either enucleated because retinoblastoma is suspected or they undergo changes which lead to serious complications and usually to loss of the eye. Of course, persistence of the hyaloid artery and persistence of the pupillary membrane are seen in adult eyes, but I have never seen, nor has any colleague of whom I have inquired seen, a single case of uncomplicated full-blown PHV in an adult.

In the literature the only identifiable cases of PHV in adults, or in those approaching adult life, are the following: (1) Ruhwandl's case.[30] The patient was 31 years old and had a partial (area 3 to 4 mm. in diameter) PHV. (2) Lane's case.[5] The patient was 14 years old and had a partial (nasal one third of the lens involved) PHV. (3) Von Grolmann's case.[11] The patient was 12 years old, and a spontaneous absorption of the lens had apparently taken place without corneal changes and without secondary glaucoma. (4) Vos's cases.[40] One patient was 20 years old. There was accompanying glaucoma for which enucleation was performed. Another patient was 17 years old. Here again glaucoma was present and an enucleation was performed. (5) Straub's case.[36] The patient was 20 years old and had a mild PHV with shrunken cataractous lens and hyaloid artery.

I have culled from the pathology collection of the Institute of Ophthalmology and the Armed Forces Institute of Pathology ten globes which had been enucleated with the clinical picture of opaque cornea, band keratopathy, and secondary glaucoma (may be buphthalmos), but which upon microscopic examination really proved to be instances of unrecognized PHV (figs. 21, 22, 23, 24). I have personally followed five cases of PHV clinically from their early, uncomplicated state to their sad cul-

mination (figs. 10, 13, 14). In the later stages the clinical picture merges into that common to other conditions (corneal scarring and glaucoma) and the correct basic diagnosis of PHV may not be made, or can be made only by surmise.

The course in cases of PHV seems invariably ill-fated for two reasons: (1) swelling of the lens due to a spontaneous rupture of the posterior capsule (see Pathology, p. 272) and (2) spontaneous hemorrhage.

The swollen and cataractous lens leads to a narrowing or an obliteration of the filtration angle (figs. 4, 7, 12, 18, 21, 22, 23, 24) and thus to secondary glaucoma. The lens, as well as the iris in front of it, protrudes towards the posterior corneal surface where if contact and adhesion occur, corneal edema and scarring ensue. If corneal contact is not made, there will be no secondary corneal changes, but there does occur complete or partial absorption of the lens with (fig. 17) or without (figs. 15, 16) secondary glaucoma. The lens may absorb, partially or completely, leaving only a membrane which may harbor calcium or capsular remains. Or, the lens may shrink to a small, smooth, white, partially calcified, globular mass.

The spontaneous hemorrhage seems to occur around the fourth month after birth and may be of a minor degree with absorption (fig. 20) and no untoward sequelae. More often, however, hemorrhage is a serious complication which augurs ill for the prognosis (figs. 13, 18, 25). The hemorrhage may be identified per se or it may be recognized by the presence of siderin in or around the retrolental tissue. Blood or its products may be appreciated inside the lens capsule or over the iris. More often than not, though, the occurrence of deep serious hemorrhage is seen only at operation when dark brown fluid and crystals exude. This deep vitreous and posterior chamber hemorrhage may lead to glaucoma, detachment of the retina, and/or eventual atrophy of the globe.

DIFFERENTIAL DIAGNOSIS

Retinoblastoma

More than any other lesion, PHV simulates retinoblastoma and is, therefore, the condition most frequently confused with it. Most of the specimens of PHV for microscopic study are available because the eyes were enucleated with a mistaken diagnosis of retinoblastoma. The differentiating points are the following: (1) Microphthalmos is not present in retinoblastoma but is usually seen to some degree in PHV. (2) PHV often manifests itself as a concave, opaque tissue just back of the lens. Retinoblastoma would never give this picture unless the vitreous were entirely filled with tumor tissue which lay against the posterior surface of the lens. (3) Eyes affected with PHV have abnormally shallow anterior chambers. Such is not the case with eyes affected with retinoblastoma. (4) PHV almost invariably manifests long ciliary processes which are visible with the ophthalmoscope or by direct illumination. Eyes affected with retinoblastoma never show such a change. (5) Eyes affected with PHV develop cataractous changes, whereas eyes affected with retinoblastoma do not.

Congenital Cataract

If a patient with PHV is seen after the lens becomes partially or totally opaque, it may be impossible to diagnose the lesion from direct observation. From inference, however, a correct diagnosis can be made when a unilateral cataract occurs in a somewhat microphthalmic globe where there is a history of development of the cataract after birth. In addition, the presence of long ciliary

processes is pathognomonic. Lastly, the presence of a very shallow anterior chamber is substantiating evidence in favor of PHV.

Judging from the histories of patients with PHV whom I have seen clinically, as well as from the histories of the patients on whom microscopic sections showing PHV are available, I find that the original diagnosis has not infrequently been congenital cataract. The cataract was attacked surgically, and enucleation was performed only after one of several subsequent developments: (1) when the fibrovascular mass was later noted back of the lens and suspected of being a retinoblastoma; (2) when the nature of the dense fibrous mass encountered back of the lens was not appreciated and therefore was not handled properly surgically, and complications such as hemorrhage and secondary glaucoma occurred; or (3) when too many unsuccessful attempts were made to effect an adequate opening in the tissue.

Retrolental Fibroplasia

It is rare for a case of retrolental fibroplasia to be confused with PHV. Retrolental fibroplasia is seldom purely unilateral. In almost every case some evidence of the sequelae of an active retrolental fibroplasia can be seen in the fellow eye at the periphery of the fundus when the patient is examined under general anesthesia. Occasionally, however, there do occur cases of retrolental fibroplasia in which no evidence of the disease can be seen in the fellow eye. Twice I have been unable to say with certainty that such cases were not instances of PHV, the difficulty being due to the fact that the affected eye in both diseases is somewhat microphthalmic, ciliary processes are visible, and the anterior chamber is shallow. Prematurity, of course, seems to indicate retrolental fibroplasia, but the disease does occur in infants with normal birth weights, and there is no reason why PHV could not occur in a premature infant. In PHV the ciliary processes are seen because they are abnormally long and incorporated in the periphery of the retrolental tissue. In retrolental fibroplasia the normal-appearing ciliary processes are seen because the contracting retrolental tissue drags the ciliary body centrally and the processes are seen in relief against the light background of the fibrous tissue. The processes are not incorporated in the periphery of the tissue. In cases of retrolental fibroplasia an accompanying cataract is almost unheard of, and when such cataractous changes do occur, they are usually in the form of localized vacuoles at the equator of the lens adjacent to the most severely involved sector of the fundus. By contrast, in PHV cataract is common and begins at the posterior pole.

There comes a time in the late stages of PHV when the clinical picture merges into that common to the late stages of other conditions. Then there is seen a partially or totally opaque cornea with or without secondary glaucoma, or with or without atrophy or phthisis of the eye. In the final stages of such ophthalmic wrecks it is sometimes impossible to state what underlying basic pathologic condition led to the terminal stage.

Retinal Dysplasia

This condition is bilateral in mentally retarded infants or in young children manifesting multiple congenital anomalies.[29]

DEPARTURES FROM THE USUAL CLINICAL PICTURE

PHV, as interpreted and depicted in this report, is a well demarcated entity. There are, however, rare cases of PHV which exhibit unusual features.

In this category is a group of cases in which there is an accentuation of the hyaloid component (figs. 28a and 28b) whereby a large stalk of vascularized tissue extends backward from the lens. These cases show varying degrees of retinal detachment and opaque white tissue around the disc. The retinal detachment is frequently seen as folds above and below the disc region, the larger fold usually being below. At other times the disc site may be obscured by white tissue which prevents a clear appraisal. The PHV element in this type of case is often minimal so that some view of the interior is possible. Three cases in this report belong to this group, and there are other such cases in the literature (Weve,[44] Wiedersheim and Herzog,[45], Snell,[34] Salffner,[31] Hervouet,[14] Hoffman,[17] Holm[18]).

Another rare allied group is characterized by the association of PHV with an iris coloboma (figs. 27a and 27b). The PHV element and usually the associated hyaloid artery are located on the posterior lens surface corresponding to the coloboma sector. One of our cases lent by Doctor Bertha Klien belongs in this category and showed a most unusual feature—the presence of melanoblasts in the retrolental fibrovascular tissue. Cases in the literature belonging to this group are those of Hess[15] (2 cases), de Vries,[43] and Eversbusch.[7]

PERSISTENCE OF THE PRIMARY VITREOUS IN OTHER CONDITIONS

In any severely malformed eye (marked microphthalmos with or without colobomas of the tunics, cyclopea, etc.), persistence of the primary vitreous with or without hyperplasia may be present. Also, in retinal dysplasia there are usually some remains of the fibrovascular sheath on the posterior surface of the lens.

TREATMENT OF PHV

On the premise that the natural course of the disease is a retrograde one, the objective of any contemplated therapy should be (1) to preserve the eye, (2) to salvage any vision that might be present, and (3) to combat any complication, such as glaucoma, that has already arisen.

It is true that eyes with PHV invariably show some esotropia (and occasionally exotropia), but this need not be considered a surgical problem of the disease.

In order to preserve the eye, the surgical objective is to handle satisfactorily the problem of the swollen lens before it produces serious and irreversible complications. In order to obtain the potential vision of the eye, an adequate opening must be made in the retrolental tissue. The problem of hemorrhage presents itself in two ways: (1) Spontaneous deep hemorrhage (fig. 25) is responsible for the loss of some of these eyes, and about this there seems to be nothing that can be done. This deep hemorrhage is appreciated at the time of surgery when old dark blood, with or without crystals and siderin, emerges from the posterior chamber. (2) Fresh, and maybe pulsating, hemorrhage may occur when the vascular fibrous sheath is cut. Usually this is moderate in amount and absorbs readily. It is surprising how negligible hemorrhage is when one considers the vascular nature of the tissue. It is true that occasionally a brisk pulsating hemorrhage occurs at the time of the discission, especially when the tissue is very thick, and this may lead to organization and secondary closure of the opening, as well as more serious complications.

The cases in this series fall into five separate surgical groups:

1. The early uncomplicated cases which should result in a good pupillary

opening after a needling followed by a discission.

II. The cases in which there has been a spontaneous absorption of the lens and only the retrolental tissue remains; no secondary glaucoma; no secondary corneal changes.

III. The cases which have shown evidence of deep spontaneous hemorrhage.

IV. The cases which have been erroneously diagnosed as congenital cataract and have been unsuccessfully operated on by the usual surgical procedures employed in these cases.

V. The cases that have had no treatment but in which the natural course of the disease has led to glaucoma.

These five groups will be separately discussed.

I. Early Uncomplicated Cases

The procedure here is to remove the lens by one or more needling operations and then to do a discission through the fibrovascular tissue. The lenses in eyes affected with PHV are rather small, and therefore, one needling usually suffices to accomplish complete absorption. The eye is ready, then, for the final discission in from four to six months' time. In these cases the posterior fibrovascular sheath varies considerably (figs. 5, 6, 7, 8) from a rather thin fibrous membrane to a tissue with a thickness even greater than that of the lens. Furthermore, this tissue may, in rare instances, contain cartilage (fig. 8). We must approach this fibrovascular tissue, therefore, with the sharpest cutting instrument available, and I know of none to compare in this regard with the Grieshaber discission knife made after the type designed by Wheeler. With such a knife, which must be exquisitely sharp, a vertical incision is made through the tissue. Because of tension from contracture, the tissue usually retracts markedly after being cut, even to the point where it lies behind the iris and leaves a round black pupil (figs. 29a and 29b). The thicker the retrolental tissue, the less it seems to retract, so that in the eyes with the thickest tissue the opening may be only a slit. The hyaloid artery enters the retrolental tissue nasal to the center so that the discission, in order to avoid the artery, should be made carefully in the center and certainly with no deviation nasally. The type of discission which has been employed is that recommended by Wheeler except that instead of one sweeping motion, several, slower, sawing motions are desirable.

Postoperatively, there is a tendency to the formation of posterior synechias, even though careful measures may have been taken to obviate them. The iris in these cases is usually rich in blood vessels, and synechias are known to form even when no surgical procedure has taken place. Especially after surgery, and particularly in the period following the needling operation, careful attention should be given to the use of mydriatics. If synechias are present when the discission is made, and if the pupil is adherent with a small diameter, the discission should include a bit of the sphincter area of the iris (fig. 30). Sometimes it seems well nigh impossible to prevent the formation of synechias.

I have treated, in the manner described above, 16 eyes belonging to Group I. Eleven of the eyes have black pupils (figs. 29, 30), the fundus details are sharply seen, and the fundi appear normal. A hyaloid artery is seen in four of the eyes. One case was a failure; the eye manifested old brown blood during the operation, indicating deep hemorrhage, and intractable glaucoma ensued. On four eyes the surgery has not been completed. (1) In one some oozing of blood covered the newly established pupillary opening and led to a thin pupillary membrane. Another discission will be necessary. (2) In another the lens

matter has been dissipated by a needling and the eye is now ready for a discission. (3) In two a needling has been done and we are now waiting for lens absorption.

From the microscopic study of eyes with PHV we know that the retina is in situ and that it apparently contains all its normal elements. Furthermore, the fovea seems well formed. Therefore, these eyes are expected to be capable of reasonably good vision. We have obtained vision as good as 15/20 in one case (fig. 30), but usually it is from 10/200 to the counting of fingers with the aphakic correction. Some of our patients are too young to cooperate. Moreover, I am sure some of the reduced vision can be attributed to disuse from obstruction of the pupillary area and the resulting tropia. Although the affected eye is somewhat smaller than its fellow eye, there seems to be no excessive amount of hyperopia present. It is my feeling that these eyes have the capacity to develop better vision through use.

II. Spontaneous Absorption of Lens with Only the Posterior Fibrovascular Sheath Remaining, and No Serious Complications Such as Glaucoma or Secondary Corneal Changes

We have had only three of these cases (figs. 15, 16) because usually complications develop which lead to loss of the eye. Two of the cases have had a discission performed in the manner described for the first group. In each case a good opening was obtained. The fundi have a normal appearance and one shows a hyaloid artery. The visual results are the same as in Group I. The third case has not had a discission to date.

III. Deep Spontaneous Hemorrhage

Hemorrhage occurring before operation may be seen as such; it may be manifested in one of its successive changes, such as siderin or crystals; or the presence of old hemorrhage may be noted at the time of operation when dark blood, siderin, or crystals, escape. Whenever there has been evidence of past deep hemorrhage in the eye, we have not had a successful outcome. There are nine cases in our series which fall into this group. Four of these cases were bilateral.

IV. Erroneous Diagnosis (Congenital Cataract)

One of our patients was said to have had a congenital cataract for which several operations had been performed. It is difficult to determine from the history exactly how many operations were done and of what nature they were. When the child was seen two years after the last operation for "congenital cataract," buphthalmos was present. The pressure was reduced to normal by a cyclodiathermy operation.

Another patient was said to have had a congenital cataract, and four operations (the nature of which was unknown) had been performed in an effort to remove the "cataract." When we saw the patient, the posterior fibrovascular sheath was present. A discission was done. This resulted in a black pupil through which the fundus appears to be normal.

V. Untreated Cases Ending in Glaucoma

This group includes six patients with PHV for which no treatment was given even though in some cases it had been advised. Subsequently, because of lens swelling, glaucoma had ensued with or without corneal scarring. Three patients in this group underwent cyclodiathermy operations and three had enucleation.

The 14 eyes which have been successfully operated on (11 in Group I, 2 in Group II, and 1 in Group IV) have shown no subsequent deterioration. In 8 of the 14 cases it has now been six

months to two years since the last operation, and in 6 it has been two to four years since the last operation. The 11 cases in Group I were probably saved from the secondary glaucoma and corneal changes which would have developed subsequent to lens swelling. The two successful cases in Group II weathered the natural hazards of the disease and were left with only the fibrovascular sheath covering an aphakic pupil. The same holds true for the one case salvaged in Group IV.

I have found only two references in the literature to a premeditated surgical plan to cope with PHV. One was by Collins,[1] who cites two cases thought to be unilateral congenital cataract in which the tissue could not be cut but was dislocated or couched. He later identified the condition as PHV and advocated needling of the lens, to be followed later by displacement or couching of the retrolental tissue. The other was by Wolfe,[46] who advocated removal of the lens and cutting or dragging the tissue free of the pupillary area.

It is possible that the surgery we have undertaken, which consisted of eliminating the lens and making an opening in the fibrovascular sheath, had no effect on whether or not the eye would suffer deep, spontaneous hemorrhage. The possibility, however, that the surgery obviated so-called spontaneous hemorrhage in some instances cannot be disregarded. We know that the fibrovascular sheath undergoes progressive contracture and that in time it becomes avascular. The constant and progressive contracture of this tissue leads to tremendous traction on the ciliary processes. These processes, as well as the ciliary body and iris, are abnormally congested with blood vessels. It is likely that this constant traction on the ciliary processes is what eventually leads to hemorrhage. The relief of this traction by the discission made through the fibrous tissue may be a factor, therefore, in preventing such hemorrhage. The fact that there has been no hemorrhage or other complications following successful surgery seems to bear this out.

SUMMARY

The clinical appearance of persistent hyperplastic primary vitreous is bizarre and sometimes unrecognized.

The clinical course, which is constantly changing, leads to complications and loss of the eye.

The eyes are enucleated because (1) of suspected retinoblastoma, (2) of intractable glaucoma, (3) of unsightly appearance from the corneal scarring and atrophy of the globe.

Frequently eyes with PHV can be salvaged by surgery.

The drawings were made by Mr. Gustave Bethke. The sections, excepting those from the Armed Forces Institute of Pathology, were prepared by Mr. Edward Gonzales.

I am indebted to the above for their help and to the Armed Forces Institute of Pathology for the use of their material which has been duly acknowledged in the legends.

Also, I am most appreciative of the help rendered by Miss Lilly Kneiske.

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LEGENDS

FIG. 1—A section of the normal eye of a 4-months fetus showing the loose vascularized fibrous tissue *a*, composing the primary vitreous. The hyaloid artery is seen at *b*. (Taken from Bach & Seefelder, Atlas zur Entwicklungsgeschichte des menschlichen Auges, 1911)

FIG. 2—A normal 7-months fetal eye, showing the character of the primary vitreous back of the lens. (Lent through the courtesy of Dr. H. C. Haden)

FIG. 3—A normal, 7-months fetal eye with the sclera resected so that the primary vitreous can be seen as a cone-shaped tissue with the base back of the lens and the apex at the disc. (Lent through the courtesy of Dr. H. C. Haden)

FIG. 4—An eye with persistent hyperplastic vitreous. The sclera has been resected to show the similarity to the primary vitreous in fetal eyes. *a*—fibrovascular tissue back of lens; *b* and *b'*—hyaloid artery; *c*—long ciliary processes; *d*—swollen lens and iris against cornea. Section of this eye shown in figure 7.

FIG. 5—A section of a globe with persistent hyperplastic vitreous. The hyaloid artery is seen posterior to the retrolental tissue. There is a rupture of the capsule of the lens at the posterior pole. The long ciliary processes extend up to the fibrous tissue.

FIG. 6—A section of an eye with persistent hyperplastic vitreous. The fibrous tissue with the hyaloid artery is shown to the nasal side of the lens and into this the long ciliary processes extend. (AFIP no. 185,415)

FIG. 7—A section of an eye with persistent hyperplastic vitreous. There is a break in the posterior capsule of the lens with adjacent cataractous changes. The lens is swollen and protrudes, cone-shaped, through the pupillary area. It touched the cornea in vivo but has retracted during preparation of the sections. Same eye as shown in figure 4.

FIG. 8—A section of an eye with persistent hyperplastic vitreous. Back of the kidney-shaped lens is an extremely thick mass of fibrous tissue in which are several areas of cartilage. Some retinal elements and ciliary processes extend up to the fibrous tissue.

FIG. 9—The clinical appearance of an eye with persistent hyperplastic vitreous. The densest part of the lesion is noted in the center. When the periphery of the tissue is viewed (right) the long ciliary processes are seen. This eye was successfully operated on and the final result is shown in figure 29-B.

FIG. 10—An eye with persistent hyperplastic vitreous which partially covers the posterior nasal surface of the lens. The densest portion is at the posterior pole. Beyond the equator of the lens nasally the long ciliary processes are seen. This eye was enucleated two and a half years later with buphthalmos and an opaque cornea. Sections of the eye show persistent hyperplastic vitreous with the hyaloid artery.

FIG. 11—An eye with persistent hyperplastic vitreous. The lens is becoming cataractous at the posterior pole. The swollen lens causes an extremely narrow anterior chamber. The insert shows the ciliary processes in the periphery nasally.

FIG. 12—An eye with persistent hyperplastic vitreous. In this eye the lens is more cataractous than the lens shown in figure 11. The swollen lens almost touches the cornea. When the pupil was miotic the iris was pushed forward, pyramidal shaped, but when dilated, as shown in the illustration, the equator of the swollen lens extended beyond the pupillary margin. The long ciliary processes can be seen indistinctly through the partially opaque lens. The iris shows many dilated blood vessels. This eye was successfully operated on and now has a round black pupil.

FIG. 13A—An eye with persistent hyperplastic vitreous showing hemorrhage around the periphery of the tissue.

FIG. 13B—The same eye four months later. Anterior synechias with corneal opacification are seen at one site; the ciliary processes have been drawn further centrally and there is an ectropion of the iris.

FIG. 14—This eye had a typical persistent hyperplastic vitreous in its uncomplicated state. The eye is shown here seven years later with anterior synechias, central corneal opacification, almost total obliteration of the anterior chamber, and a band keratopathy.

FIG. 15—This eye has a persistent hyperplastic vitreous with spontaneous absorption of the lens. The yellowish island of tissue is the only lenticular remains seen. The ciliary processes are noted around the periphery. There is no glaucoma. The eye was successfully operated on.

FIG. 16—An eye with persistent hyperplastic vitreous. The lens has been completely absorbed. The only lenticular remain seen is the white island of tissue above. The fibrous tissue has undergone contracture and pulled the ciliary processes far centrally. No glaucoma is present.

FIG. 17A—An eye with persistent hyperplastic vitreous. The lens has undergone spontaneous absorption except for a translucent shrunken capsule in the upper part of the pupillary area. The iris is rich in blood vessels and these extend onto the fibrous tissue covering the pupillary area. There was intractible secondary glaucoma and the eye was enucleated.

FIG. 17B—The enucleated globe showing the hyaloid artery which could be traced to the disc. The lenticular remains shown in 17A can be seen here in the pupillary area. The angle is obliterated by synechias.

FIG. 18—A case of bilateral persistent hyperplastic vitreous. A—Right eye: Hemorrhage can be seen around the periphery of the fibrous tissue. B—Left eye: There are anterior synechias, corneal opacification, and secondary glaucoma.

FIG. 19—A case of bilateral persistent hyperplastic vitreous. In each eye the lens is shrunken to a small globular mass. Behind this is a fibrous tissue around the periphery of which are the ciliary processes. There is ectropion of the pigment epithelium of the iris.

FIG. 20A—A mild case of persistent hyperplastic vitreous followed for 14 years. There is an island of fibrovascular tissue on the posterior surface of the lens nasally. Several ciliary processes can be seen just inside of the pupillary margin. The patient was 4 days old. At the age of 4 months a hemorrhage occurred over the surface of the fibrovascular tissue. The hemorrhage gradually absorbed.

FIG. 20B—The same eye shown in 20A, 14 years later. The fibrous tissue has contracted and pulled the ciliary processes more centrally where they are seen between the equator of the lens and the pupillary margin. The lens is partially cataractous adjacent to the fibrous tissue.

FIG. 21—An eye with persistent hyperplastic vitreous enucleated because it became blind, uncomfortable, and disfiguring. The partially absorbed lens and iris are in contact with the cornea. Back of the lens is a fibrovascular sheath into which the ciliary artery enters. There are peripheral synechias. The presence of persistent hyperplastic vitreous was not appreciated before enucleation as the basic cause of the disease. (AFIP no. 188,158)

FIG. 22—An eye with persistent hyperplastic vitreous enucleated with a clinical diagnosis of advanced buphthalmos. There is a partially absorbed lens, back of which is a fibrovascular sheath with long ciliary processes inserting into it. The iris is against the posterior surface of the cornea. There are secondary corneal

changes. The hyaloid artery was present in other sections.

FIG. 23—An eye with persistent hyperplastic vitreous enucleated because it was blind, uncomfortable, and disfiguring. There is an almost completely absorbed lens, back of which is the fibrovascular sheath with the hyaloid artery inserting into it. The anterior chamber is obliterated and there are secondary corneal changes. Persistent hyperplastic vitreous was not suspected before enucleation. (AFIP no. 96,585)

FIG. 24—A blind disfiguring eye not suspected of having persistent hyperplastic vitreous before enucleation. The partially absorbed lens is adherent to the posterior surface of the cornea. There is a thick fibrovascular tissue into which the hyaloid artery inserts. (AFIP no. 211,920)

FIG. 25—An eye enucleated because it was a retinoblastoma suspect. There is evidence of old hemorrhage with organization and contracture. Back of the lens is persistent hyperplastic vitreous occupying the funnel of the completely detached retina.

FIG. 26—An eye with persistent hyperplastic vitreous enucleated because it was a retinoblastoma suspect. There is complete absorption of the lens with only the fibrovascular tissue remaining. The hyaloid artery can be seen coursing for a short distance posterior to the tissue. (AFIP no. 169,162)

FIG. 27—Persistent hyperplastic vitreous associated with a coloboma of the iris and other congenital anomalies. The left eye of a colored infant who died at the age of 2 months. There were multiple congenital anomalies throughout the body. A—A section at one level to show the posterior fibrovascular sheath with the hyaloid artery inserting into it. B—A section at another level to show this tissue extending through an iris coloboma to the posterior surface of the cornea. The fibrovascular sheath shows densely pigmented melanoblasts. (Specimen lent through the kindness of Dr. Bertha A. Klien)

FIG. 28—Cases in which the hyaloid component predominates. A—A small disc of opaque tissue is seen just posterior to the lens, and from this the hyaloid artery courses toward the disc. A vertical retinal fold extends across the disc. B—Section of an eye with a lesion similar to that seen in (A). (AFIP no. 543, 207)

FIG. 29—(A) An eye with persistent hyperplastic vitreous after almost complete disappearance of the lens following a needling. Around the periphery of the fibrovascular tissue are the ciliary processes. This is the appearance of the eye just before the discission was made. (B) The appearance of the eye after the discission showing that the fibrous tissue retracted to such a degree that a round black pupil is present. The fundus is normal except for the hyaloid artery which is depicted in the drawing extending from the disc to the nasal part of the fibrous tissue which has retracted back of the pupil. The clinical appearance of this eye before surgery is shown in figure 9.

FIG. 30—An eye with persistent hyperplastic vitreous after surgery. The insert shows the vertically oval black pupil. From the nasal side of the pupil the hyaloid artery extends posteriorly to the disc. Otherwise the fundus is normal. Vision, 15/20 with aphakic correction.

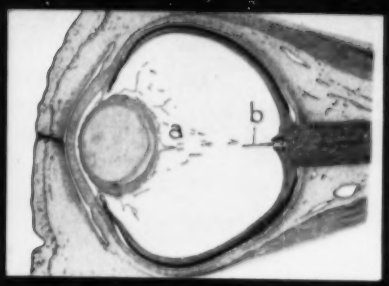
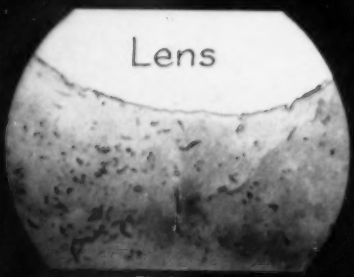


Figure 1



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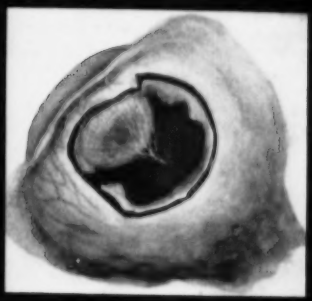

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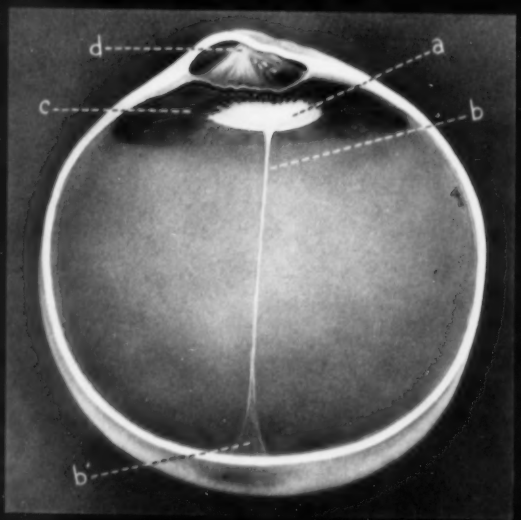

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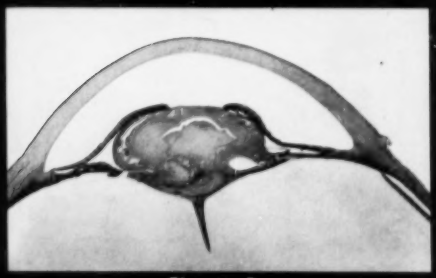

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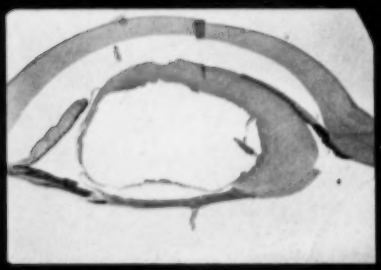

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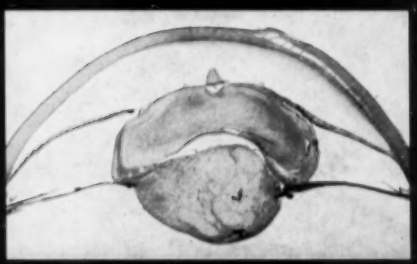
Figure 7



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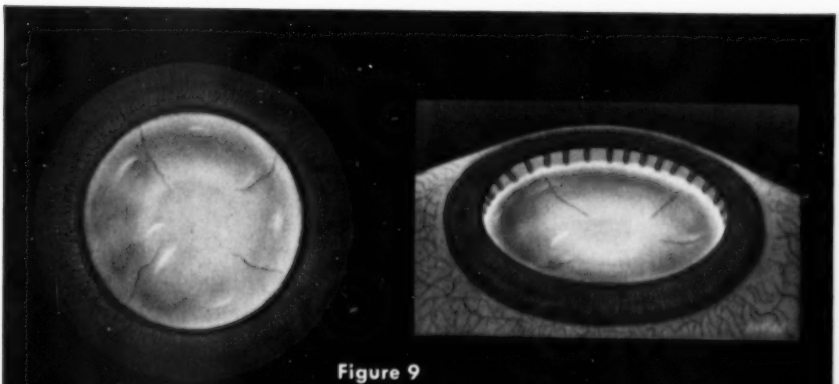

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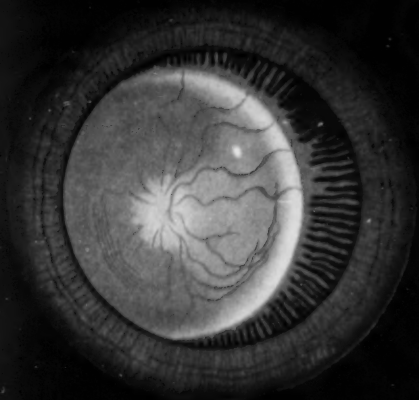

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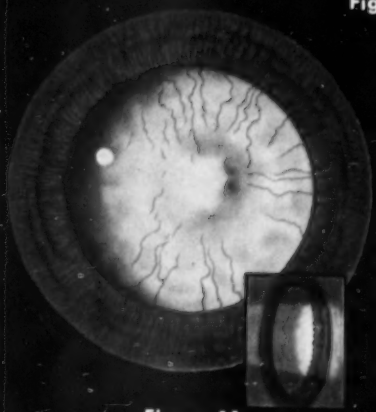

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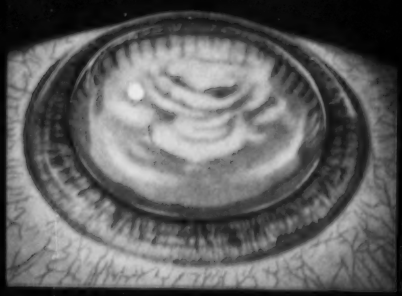

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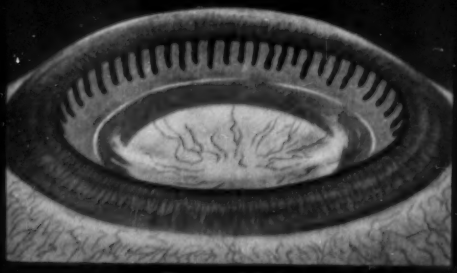


Figure 13 A

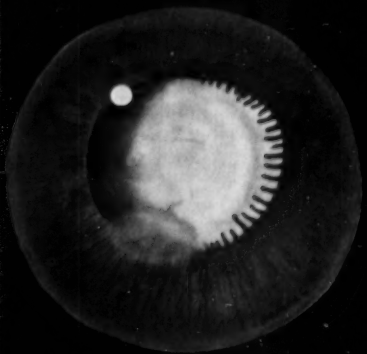
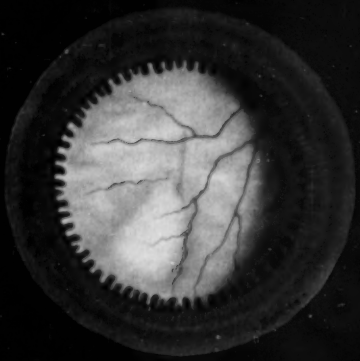


Figure 13 B



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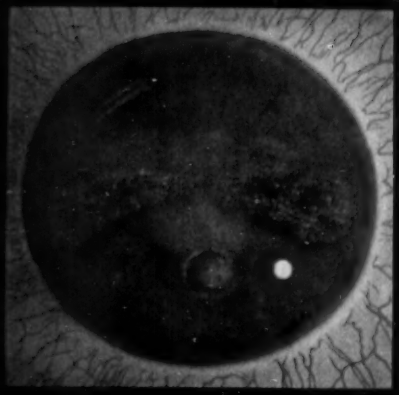


Figure 14

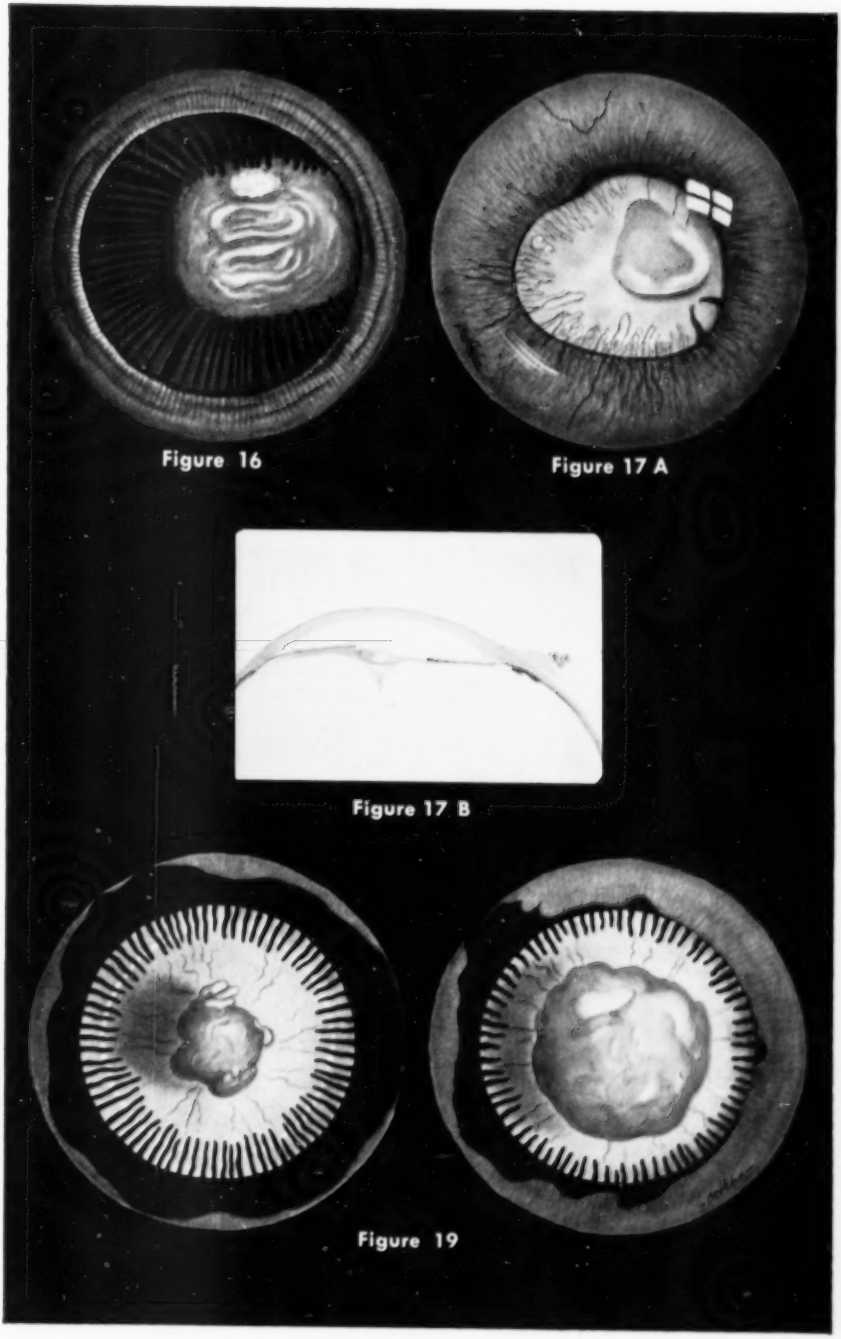


Figure 16

Figure 17 A

Figure 17 B

Figure 19

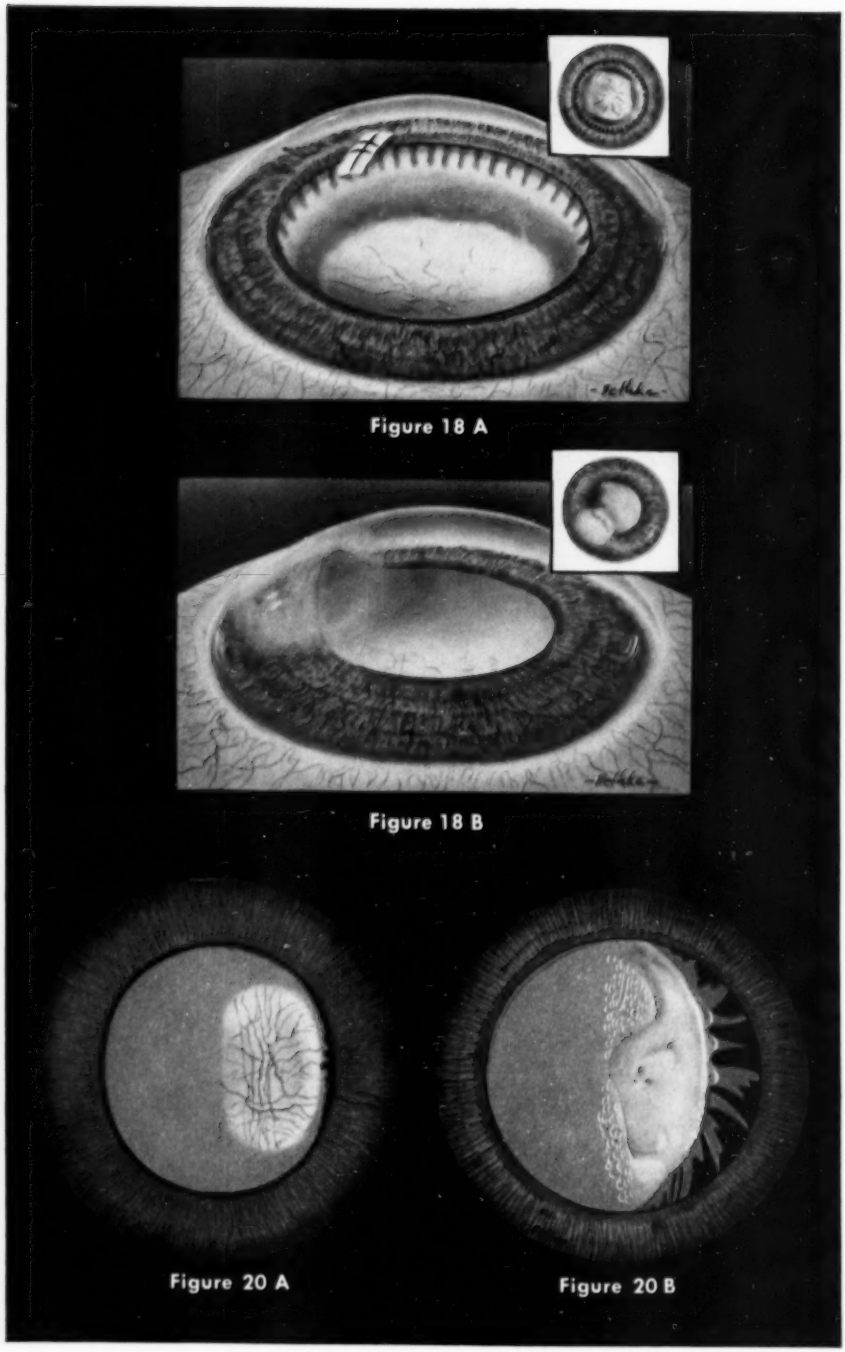


Figure 18 A

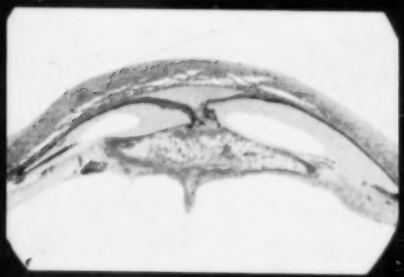
Figure 18 B

Figure 20 A

Figure 20 B

Figure 21

Figure 22



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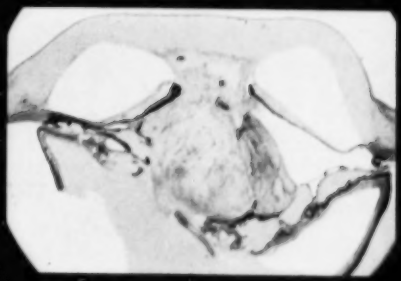

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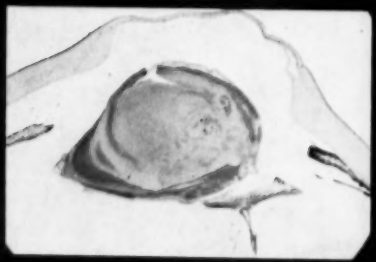

Figure 27 A

Figure 27 B

Figure 28 B

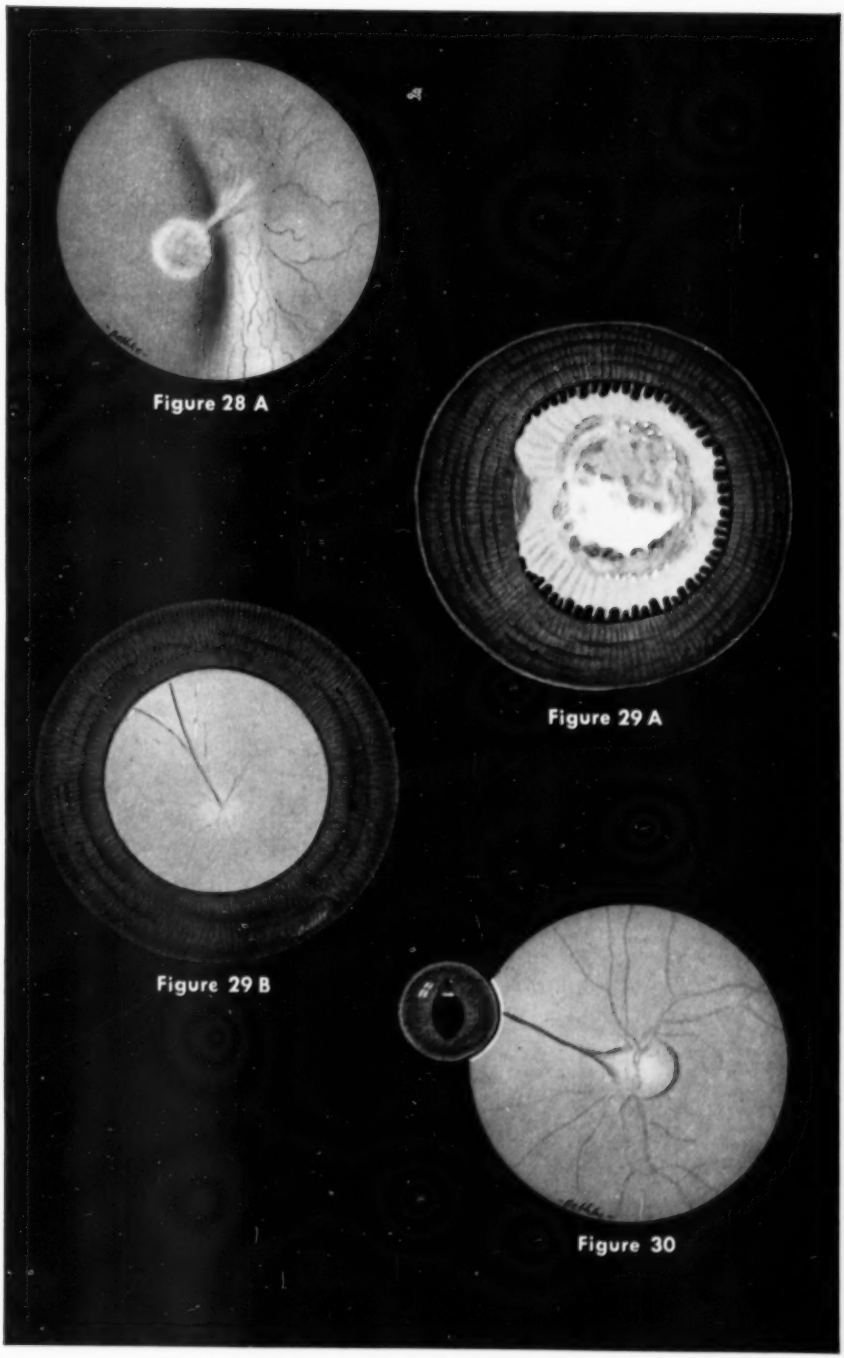

Figure 28 A

Figure 29 A

Figure 29 B

Figure 30

Symposium: Congenital Glaucoma

PATHOGENESIS OF CONGENITAL GLAUCOMA: GONIOSCOPIC AND MICROSCOPIC ANATOMY

ROBERT N. SHAFFER, M.D.
SAN FRANCISCO, CALIFORNIA

THE term "congenital glaucoma" applies to those cases of increased intraocular pressure in which the decreased aqueous outflow is due to defective embryologic development of the eye. In one group of these cases, symptoms are present at birth or appear within the first three years of life. These are known as infantile glaucoma. At this age, the ocular structures are so elastic that increased intraocular pressure results in the distended eyeball known in bygone years by the picturesque terms "buphthalmos" and "hydrophthalmia."

After the age of three, the cornea and sclera become more resistant to stretching, and if the intraocular tension rises, a chronic simple type of glaucoma results which merges into the adult or senile form of open angle glaucoma.[24] This is called juvenile glaucoma. Since it may occur between the ages of three and thirty-five, cases of this type do not form an etiologically pure group.

JUVENILE GLAUCOMA

Although there are reports in the literature of children with infantile glaucoma whose siblings were afflicted with the juvenile variety,[21,25,28] and there are other papers which state that the angle anomaly is the same in infantile and in juvenile glaucoma,[1,13] it seems to the members of this symposium that infantile glaucoma and the juvenile glaucomas represent separate entities. It is known that faulty genes may cause developmental defects for some time after birth. As a result, there may be confusion in diagnosis in the first decade between a juvenile glaucoma and an infantile glaucoma which has had either a delayed onset or a spontaneous resolution. Similarly, in the period from the second to fourth decade of the patient's life it becomes increasingly difficult to separate the congenital from the degenerative disease. In this age group there are eyes with a narrow angle which may have typical attacks of acute glaucoma. There are other cases which are indistinguishable from the open angle glaucoma of the adult. No dogmatic statement can be made as to whether these should be considered as congenital in origin or as the premature development of a degenerative process.

The true congenital juvenile glaucoma may present no angle anomaly visible gonioscopically to explain the condition. In some cases the trabecula may be longer than usual and the line of Schwalbe quite prominent. However, similar findings have been noted in 15 per cent of routine eye examinations by Burian, Braley and Allen.[9] Iris processes, small and large, or entire sheets of iris stroma may circle the angle recess, extending in a variable pattern and for varying distances over the trabecula. The more

From the Division of Ophthalmology, University of California Medical School, San Francisco, Calif.
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severe forms of this phenomenon make up the syndrome of Axenfeld,[3] or posterior embryotoxon, which is inherited in a dominant pattern, according to Falls.[14] The milder anomalies may escape clinical note but should be grouped under the same heading, as suggested by Streiff.[29] Increased intraocular tension may never become a problem in these cases, or it may appear as late as the fifth decade of life.

Pigmentary glaucoma, described by Sugar[30] and Calhoun,[11] must also be considered in the differential diagnosis of any young person with increased intraocular tension. However it is improbable that it can be considered a congenital glaucoma.

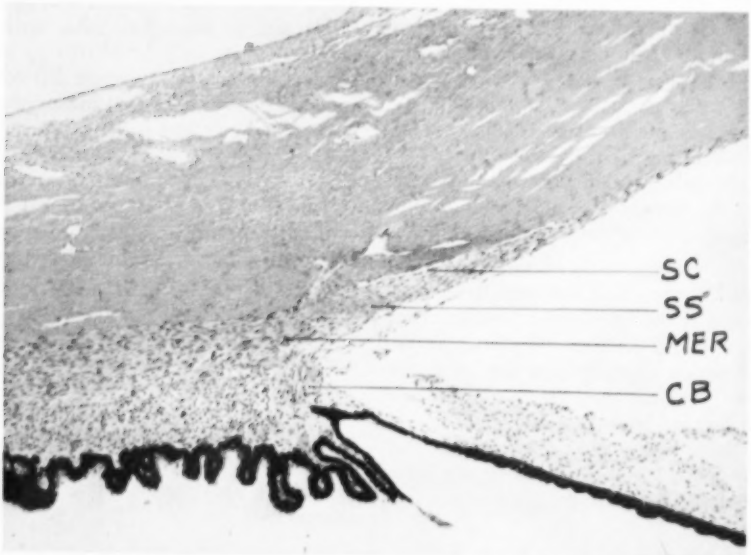

FIG. 1—Anterior chamber angle of normal infant eye. SC—Schlemm's canal; SS—scleral spur; MER—meridional fibers; CB—ciliary body. (Shaffer)

GLAUCOMA ASSOCIATED WITH OTHER CONGENITAL ANOMALIES

Aniridia, or irideremia, is inherited as a dominant characteristic.[15] The rim of remaining iris is usually visible only by gonioscopy. The stump of iris is lifted up against the trabecular area, sometimes nearly to Schwalbe's line. Often the onset of the increased tension is delayed into adult life.

Nevus flammeus, or the Sturge-Weber syndrome, is a congenital condition which is usually unilateral. It is frequently complicated by increased intraocular tension which may be present at birth, but the elevation may not appear for several years. The angle looks somewhat like that in infantile glaucoma, though some cases have greater vascularity of the iris and angle. There is often an associated angioma of the choroid and of the brain. Since Barkan has reported that goniotomy may be effective in controlling the glaucoma in both aniridia[6] and in nevus flammeus,[7] it seems probable that the angle anomaly is the cause of the glaucoma.

Neurofibromatosis is an heredofamilial disease in which the glaucoma is always unilateral, as are the facial

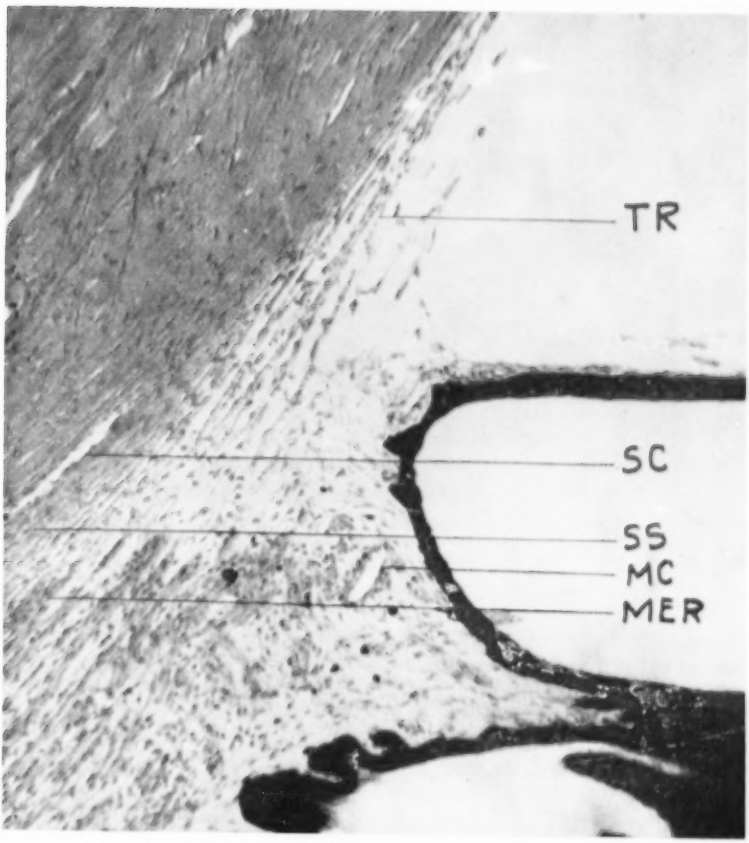

FIG. 2—Anterior chamber angle of eye with infantile glaucoma. TR—trabecula; SC—Schlemm's canal; SS—scleral spur; MC—major circle; MER—meridional fibers. (Maumenee)

changes. Anderson states that hypertrophied ciliary nerves and a diffuse thickening of the choroid appear to be constant changes.[2] The characteristic change in the angle is the presence of an iris-like sheet of tissue running up on the trabecula.

INFANTILE GLAUCOMA

True, primary infantile glaucoma is caused by anomalies of ocular development in the fetal period. It is conceivable that agents other than defective genes might be responsible. Illness of the mother from such conditions as rubella,[18] lues, toxemia, or hypoxia, could play a part. In general, however, the evidence indicates its inheritance as an autosomal recessive characteristic, according to Falls and to Franceschetti.[16] This belief is supported by the multiple occurrence of the disease in the offspring of consanguineous parents.[15] Two thirds of the cases occur in boys, and a similar proportion are bilateral. Other anomalies, such as congenital heart disease, deafness, mental deficiency and pyloric stenosis, may coexist.

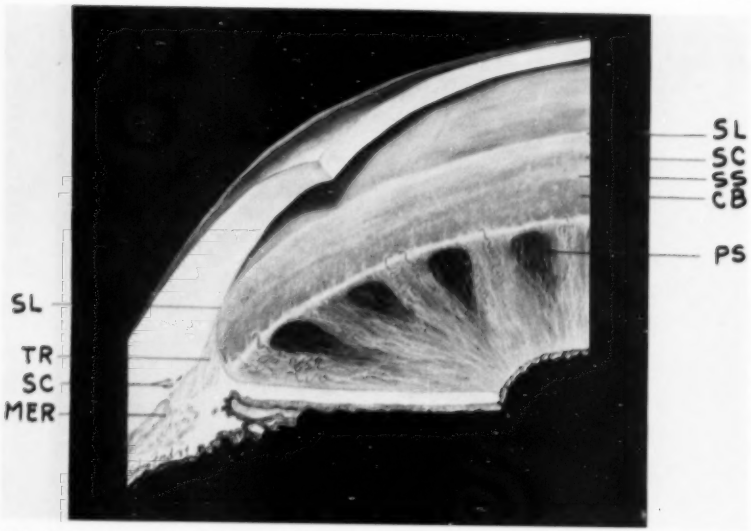

FIG. 3—Composite drawing of angle in infantile glaucoma, showing microscopic and gonioscopic appearance. SL—Schwalbe's line; SC—Schlemm's canal; SS—scleral spur; CB—ciliary body; PS—pigment sectors; MER—meridional fibers. (Shaffer)

Pathologic and Gonioscopic Anatomy

The vast majority of anatomic specimens available to the pathologist is made up of badly damaged eyes. After the failure of numerous operations, enucleation has been performed because the eyes were blind, painful, or cosmetically disfiguring. These distended eyes are pathologic museums, but in themselves can offer little evidence of the primary defect responsible for the increase in intraocular tension. It is of interest, however, that changes similar to those of early specimens are seen in these large eyes when they have not been too badly damaged. Pathologic changes of late infantile glaucoma will not be considered here.

In this paper, study will be limited to the gonioscopic anatomy of early cases and to the pathologic microscopic anatomy of eyes which have had little or no surgical interference, and which have suffered no greater distension than that represented by a corneal diameter of 14 mm. Until recently, the only eyes of this type in which the microscopic anatomy has been reported have been those summarized by Anderson[2] in his monumental work. A maximum of fifteen such cases is presented. One of these had a partial aniridia, and another neurofibromatosis. In recent years Dr. A. Edward Maumenee has collected fifteen eyes from various sources, which fulfill the same criteria. Members of this panel are indebted to him for his permission to study his cases, and for his help in interpreting the pathologic changes.[22]

A. The Iris

In normal infant eyes at birth, the angle recess is not yet well developed (fig. 1). Some of these angles can resemble to some extent, the changes seen in infantile glaucoma. However, in the latter, the iris can be seen microscopically to insert directly into the trabecula,

anterior to the spur, instead of onto the ciliary body as in the normal eye (fig. 1). This high insertion usually covers one fifth to one third of the trabecula, but in rare cases over one half of the trabecula may be covered (fig. 2). Such a block to aqueous outflow could be responsible for the increased tension of infantile glaucoma.

The gonioscopic findings, first described by Otto Barkan,[7] match the microscopic findings to a considerable extent (fig. 3). There is no angle recess, nor insertion of iris on the ciliary body. The anterior iris leaf is not well developed. In blue-eyed babies, delicate gray-blue strands of connective tissue run radially toward the angle. Through this semitransparent tissue, as it nears the angle, the pigment layer of the iris can be seen in arcade-like sectors. Variations of this picture are also seen in normal babies. As the gray strands and radial blood vessels reach the inner surface of the trabecular wall, they rise, or are slightly picked up on the translucent tissue of the angle. The pigment sectors beneath this surface stroma also appear to be lifted peripherally. In some instances the anterior stroma sweeps up quite high toward Schwalbe's line. This forms the "hook-like process" described by Seefelder[27] (fig. 4), and is continuous with the uveal portion of the trabecula.[31] There is usually a definite recession of the anteriorly placed iris and pigment sectors in the area of a successful goniotomy.

B. The Scleral Spur and Ciliary Body

The scleral spur is much less prominent in the eye with infantile glaucoma than in the normal eye and is invariably well behind the angle recess in the microscopic sections. It often can be identified only by the insertion point of the radial muscle fibers of the ciliary body (fig. 2). This condition is quite unlike

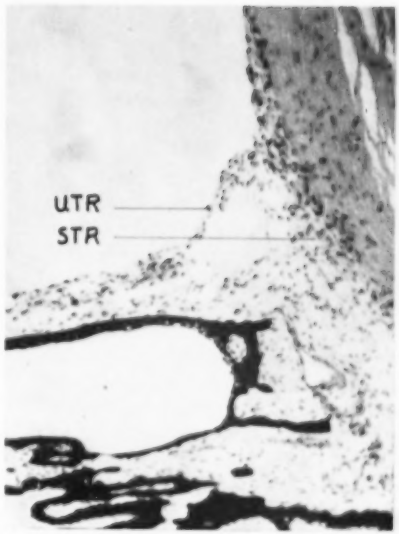

FIG. 4—Infantile glaucoma, showing "hook-like process" of Seefelder. UTR—uveal trabecula; STR—scleral trabecula. (Shaffer)

that in the normal infant's eye, in which the spur is seen at the level of the angle in most instances. The ciliary body in the glaucomatous eye is atrophic and poorly developed. This may offer an explanation for the delayed onset of increased tension. It is possible that aqueous is being secreted at such a low rate that pressure fails to build up at first. When inflow begins to approach normal, the defective outflow channels cause a rapid increase in tension.

Gonioscopically, the ciliary body is seen as a shadowy gray area. It is seen through the anterior iris stroma beyond and beneath its attachment to the trabecula. A white area is seen just anterior to it; it looks like the scleral spur in the adult, though it may merely be scleral tissue shining through the semitransparent trabecular tissues. When there is blood in Schlemm's canal, it is seen definitely anterior to this white zone (fig. 3).

C. *The Trabecula*

Seefelder[27] states that the inner lamellas of the trabecula are more compressed in infantile glaucoma than when the eyes are normal. He also points out an abnormal insertion of the trabecula into the ciliary body rather than into the scleral spur. This seems to be true also of many of the specimens studied by the present panel (fig. 5). However, when comparing photomicrographs of the trabecula of glaucomatous and of normal infant eyes, no consistent differences were seen. In addition to the variability in the degree of development of the angle at birth, the appearance of the trabecula is changed considerably by slight variations in the thickness and direction of the microscopic sections. Its thickness, the number of nuclei present, and the size of the trabecular fibers are variable and can be misleading. Although definite conclusions cannot be made from the microscopic appearance of the trabecula, it does seem probable on the basis of the clinical findings and the results of goniotomy that there is some defect in the permeability of this tissue. Certainly in some pathologic specimens and eyes seen gonioscopically, the anterior iris insertions are not high enough in themselves to stop filtration (fig. 6).

Gonioscopically, the trabecular wall is more transparent than in the adult. Otto Barkan[7] believes it is not as transparent as in the normal infant. This surface has a definite shagreen as seen by oblique illumination, looking almost like stippled cellophane. It is continuous with the anterior iris stroma and probably represents part of the uveal portion of the trabecula. The thick translucent tissue behind it is the bulk of the corneoscleral trabecular fibers. Gray fibrils of anterior iris stroma with radial blood vessels can be seen lifted up on its surface. Some of them turn back down through the semitransparent trabecula toward the ciliary body (fig. 3). Barkan states that the angle wall is more vertical than with a normal trabecula. He believes that this has no relation to the corneal enlargement of these cases.

Many authors have spoken of the mesoblastic reticulum which fills the angle of the eye with infantile glaucoma.[5,17] Kluyskens[19] states that there is no high insertion of the iris. He thinks that the iris is normally placed, but that the abnormal mesodermal tissue lying on top of it gives the gonioscopic appearance of a high insertion.

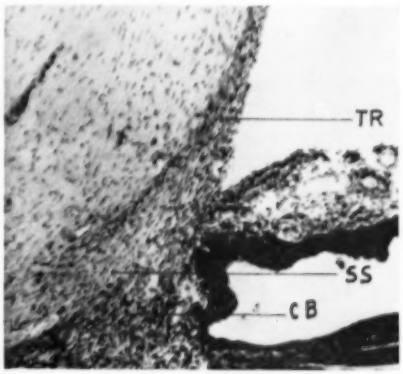

FIG. 5—Infantile glaucoma. TR—trabecula; SS—scleral spur; CB—ciliary body. (Maumenee)

There is no doubt that there is a semitransparent membrane veiling the peripheral anterior iris and continuing up over the inner trabecula. From our studies we have come to believe that this represents the uveal portion of the trabecula and not an abnormal mesoblastic tissue occupying space in an otherwise normal angle. The trabecula itself may be anomalously differentiated to account for its relative impermeability. Treacher Collins,[12] Seefelder,[27] and more recently, Burian[8,9] and Maumenee,[23] have suggested that the angle develops by actual cleavage of the tissues rather than by absorption of mesoblastic remains. If so, the angle defect could

FIG. 6—Infantile glaucoma. SC—Schlemm's canal; AI—anterior insertion; MC—major circle. (Maumenee)

well be explained on the basis of incomplete cleavage of iris tissue from posterior cornea. It may also be possible that goniotomy is effective by furthering that cleavage.

D. *Schlemm's Canal*

Schlemm's canal is poorly formed for the most part and cannot be recognized easily in the sectioned eyes (fig. 7). In the living eye, with congenital glaucoma, a red band which looks like a blood-filled Schlemm's canal has been described.[4,10] Although the microscopic sections show only irregular endothelial channels, by gonioscopy it is usually possible to observe a blood-filled channel approximately identical in position and appearance to that of a blood-filled Schlemm's canal in the adult and in normal infants, produced by jugular compression or by aqueous aspiration, as suggested by Scheie.[26] This channel is often somewhat irregular in size and caliber, and varies in color from a faint pink to a brilliant red. Despite the microscopic evidence that the rudimentary canal should be hidden behind the anterior iris insertions, the red band is usually well anterior to the point of iris insertion. Some eyes have been examined in which a variable line of the iris insertion permitted a blood-filled Schlemm's canal to be seen in some areas and to be concealed in others (fig. 8). This discrepancy is easily seen by comparing figure 3 with figure 2.

It has been suggested that the red line may represent an increased vascularity of the intrascleral venous plexus when the jugular veins are compressed and that the effect of goniotomy in lowering

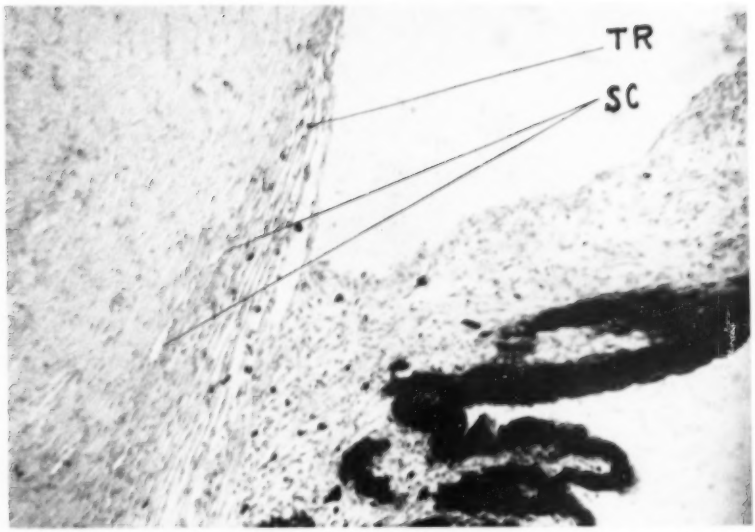

FIG. 7—Infantile glaucoma. TR—trabecula; SC—Schlemm's canal. (Maumenee)

tension may be due to an incision into the sclera permitting access and absorption of aqueous by the intrascleral venous plexus.[22] It is hard to believe that additional blood in the intrascleral venous plexus could give such a perfect picture of the size, shape, color and position of a normal Schlemm's canal. If the tension is elevated, the red streak does not usually appear unless a paracentesis of the chamber is done. This would seem to indicate that the intraocular tension might be holding shut potentially open channels. When the tension is normalized by goniotomy, it is then easy to fill this channel with blood, just as it is easy to fill a similar canal in the normal infant eye. It is easier to conceive of increased intraocular pressure pressing upon the impermeable trabecula and holding closed a potentially open Schlemm's canal than to think of that pressure holding blood out of a deep venous plexus.

After incision of the trabecula by goniotomy there is often a surprising normalization of tension, even when only one quadrant of the angle has been operated upon. The coefficient of aqueous outflow has been found to be increased from below 0.10 to over 0.20 by Kronfeld[20] and by members of the panel. This indicates that drainage channels are of more than adequate capacity.[7]

Etiology

In view of the findings which have been presented, what can be said as to the probable etiology of infantile glaucoma? There is no argument as to its recessive inheritance, nor that some type of anomaly in development is responsible for the glaucoma. Kluyskens[19] and Weekers and Weekers[32] state that the angle lesions are insufficient to explain the increased tension, and they suggest that it is due to an attendant de-

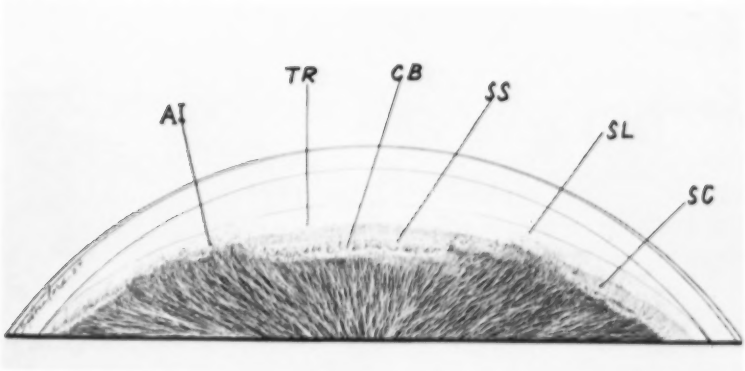

FIG. 8—Angle in infantile glaucoma showing relation of Schlemm's canal to anterior iris insertions. AI—anterior insertion; TR—trabecula; CB—ciliary body; SS—scleral spur; SL—Schwalbe's line; SC—Schlemm's canal. (Shaffer)

velopmental anomaly of the uveal vascular system. No evidence of such an anomaly has been found. The success of goniotomy in treating these cases is an argument against this theory, as a superficial incision of the trabecula could hardly alter neurovascular factors to any material extent.

This panel believes that the block to aqueous outflow is at the trabecular level. Tension may be normal at birth because the underdeveloped ciliary body is producing aqueous at a low rate. As inflow begins to approach normal, the block to outflow causes a rapid increase in intraocular tension, and symptoms appear. What can be said as to the nature of this block?

As seen microscopically, one would be certain that the block was caused, first, by the high insertion of the iris on the trabecula and, second, by the poor development of Schlemm's canal. As seen gonioscopically, one is surprised to find that in most cases Schlemm's canal can be filled with blood, and that this red band can be seen in the angle wall, well above the apparent point of attachment of iris to trabecula. Since these microscopic and gonioscopic aspects are based upon the same anomaly, any discrepancy between the two must be due to the distortions of tissue and of vision inherent in each method.

It seems probable that embryologically there is an incomplete separation of iris tissue from posterior cornea. This results in the iris partially covering the more permeable posterior half of the trabecular meshwork. This adds a variable amount of block depending upon the height of that insertion. There is no evidence of an abnormal unresorbed mesoblastic reticulum filling the angle, as has been described in the past. There is probably, also, a relative impermeability of the trabecula. Goniotomy incises this impermeable trabecula (figs. 9, 10). The same incision causes a recession of the iris, which drops back as it loses the support of the trabecula. In the majority of cases, an adequate Schlemm's canal is available if an incision can be made in the right area. There are undoubtedly some cases in which the canal is so faulty that goniotomy is doomed to failure.

Although no successfully gonioto-

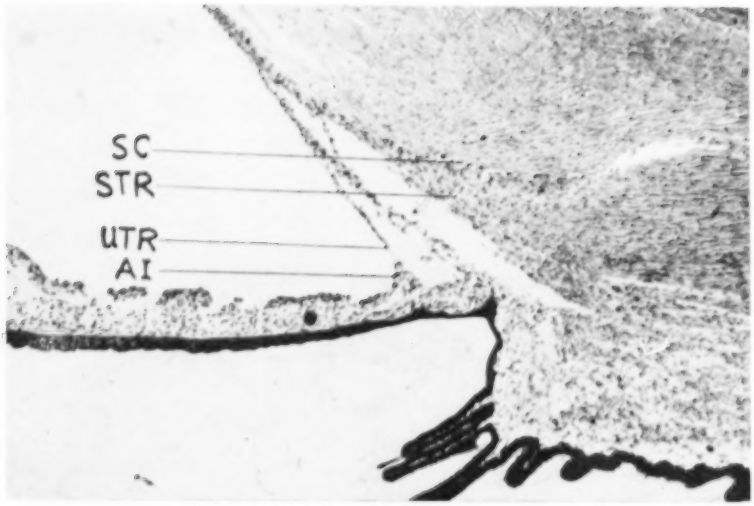

FIG. 9—Unopened right eye in case of infantile glaucoma. SC—Schlemm's canal; STR—scleral trabecula; UTR—uveal trabecula; AI—Anterior iris insertion. (Christensen)

mized eyes have been examined microscopically, it seems probable that endothelium lines the incision and maintains its integrity. If the incision is made too deeply into the sclera, failure may result from closure of the opening by fibroblastic proliferation before it can be endothelialized. In neglected cases, the stretching caused by an advanced glaucoma, or damage by repeated surgery, results in extensive peripheral anterior synechias and permanent closure of the canal of Schlemm. For this reason, goniotomy is ineffective in late infantile glaucoma.

A great deal more work needs to be done before the pathogenesis of infantile glaucoma can be said to be established. If the trabecula is truly impermeable, as is suggested, the nature of this alteration should be investigated. The absence of blood in Schlemm's canal before normalization of pressure, and its presence afterwards, is an intriguing problem. It is hoped that this discussion may serve as a starting point for more basic research in the future.

SUMMARY

1. A short description of the various types of juvenile glaucoma is presented.
2. The microscopic anatomy of infantile glaucoma shows no abnormal mesodermal reticulum. There is a high insertion of the iris on the trabecula. Schlemm's canal is not perfectly formed.
3. It has been demonstrated gonioscopically that Schlemm's canal can be filled with blood in most cases of infantile glaucoma. There is a direct insertion of iris into trabecula, but it is not as high as is apparent in the microscopic sections. There are some clinical findings which suggest decreased trabecular permeability.

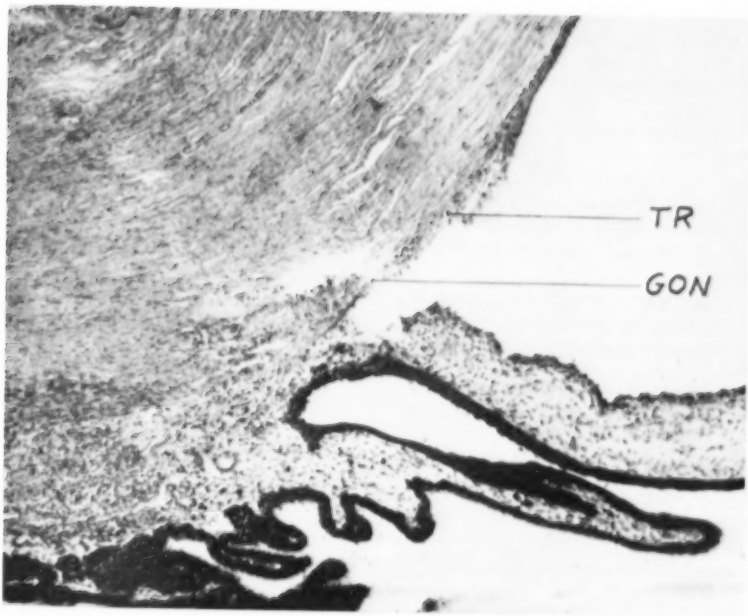

FIG. 10—Left eye in case of infantile glaucoma operated upon by goniotomy. Cardiac arrest following surgery. TR—trabecula; GON—goniotomy incision through trabecula with slight penetration into sclera. (Christensen)

4. It is suggested that the etiology of infantile glaucoma is due in part to the anomalous insertion of iris into the most permeable part of the trabecula, and in part to some impermeability of the trabecula itself. In an occasional case, Schlemm's canal may be too faulty for goniotomy to be successful.

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DIAGNOSIS, CLINICAL COURSE, AND TREATMENT OTHER THAN GONIOTOMY

HAROLD G. SCHEIE, M.D.
PHILADELPHIA, PENNSYLVANIA

THE diagnosis of infantile glaucoma presents few problems when the classical clinical picture is fully developed. However, when the patient is seen very early in the disease and the classical findings are absent, the difficulties of diagnosis can be as great as in primary glaucoma of the adult. The importance of early diagnosis is emphasized by the excellent response of infantile glaucoma to modern surgical methods when they are employed early in the disease and the very poor response when the disease is advanced. Ophthalmologists, pediatricians and obstetricians, therefore, should be constantly on the alert for its presence.

The importance of infantile glaucoma has been emphasized by numerous authors. Anderson,[3] in reviewing statistics from various eye clinics, found the incidence to vary from 0.03 per cent to 0.079 per cent of all clinic patients. Lehrfeld and Reber[24] found that it occurred in 0.011 per cent of the 250,000 patients attending the clinics at Wills Eye Hospital from 1926 to 1935. Anderson,[3] in his survey, noted that infantile glaucoma as a cause of blindness varied from 2.4 per cent to 13.5 per cent of the children in institutes for the blind. Bailliart[4] reported that infantile glaucoma was the cause of blindness of 23 per cent. Westerlund[53] found an incidence of 6.4 per cent in different schools for the blind.

From the Department of Ophthalmology, Hospital of the University of Pennsylvania, Philadelphia, Pa., and The Children's Hospital of Philadelphia.

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AGE OF ONSET

Signs of infantile glaucoma may or may not be present at birth. In many instances, the exact time of onset is difficult to determine. Irritative symptoms may not appear until sometime after birth, but the parents, not infrequently, comment upon how large and beautiful the child's eyes have been since birth. In my own series of 45 patients, the diagnosis was apparent at birth in 14. By six months of age, the diagnosis had become apparent in 36 of the 45 patients. In the remainder, the onset could not be definitely determined, but in most cases, it was apparent by the time the child was one year old. In Barkan's series of 87 eyes,[7] the corneas of 30 were cloudy at birth. Gros[21] noted that in 45 eyes, the disease was present at birth in 60 per cent. Of the remainder, it was detected in 13.3 per cent during the first year; in 17.7 per cent between the first and third years; and in 8.8 per cent subsequent to that time. Anderson,[3] in summarizing papers by Seefelder, Dettmering, Stolting, and Fleischer, quoted the following statistics:

Present at or before birth in 102 (40%)
Present at 6/12 months or under in 86 (34%)
Present at 1 year or under in 30 (12%)
Present at 6 years or under in 28 (11%)
Present at over 6 years in 5 (2%)
Unreliable or no information in 80

Not only may the signs of infantile glaucoma be present at birth, but the disease may be very advanced. However, the very significant number of patients born with no signs or symptoms of the disease emphasizes the importance of being constantly on the alert for the condition, particularly during the first year of a child's life.

SYMPTOMATOLOGY

The earliest symptoms of the disease are photophobia, blepharospasm, and epiphora. Any child who is less than a year old and who has these symptoms should be considered to have infantile glaucoma until this diagnosis can be excluded. This is true whether or not haziness of the cornea, enlargement of the cornea, and other clinical signs are present. In my own experience, photophobia is usually the first manifestation of the disease in those eyes where the onset is after birth and the entire sequence of the disease can be observed. The mother often states that the child first became sensitive to light, and that the other signs of infantile glaucoma developed later.

Photophobia also is an excellent criterion for success or failure of treatment because once the tension is controlled, this symptom usually subsides promptly. On the other hand, continued photophobia nearly always indicates persistently elevated ocular tension even though the cornea may have cleared.

Objective Signs

The classical objective signs of infantile glaucoma consist of corneal haze (fig. 1), enlargement of the globe with increase in corneal diameter, ruptures of Descemet's membrane (fig. 2), and deepening of the anterior chamber. These signs all result from the fact that infantile glaucoma occurs at an age when the ocular coats are sufficiently pliable to stretch under increased intraocular pressure. A corneal diameter of more than 12 mm. should always be regarded with suspicion, and a corneal diameter of even less than this size is no assurance

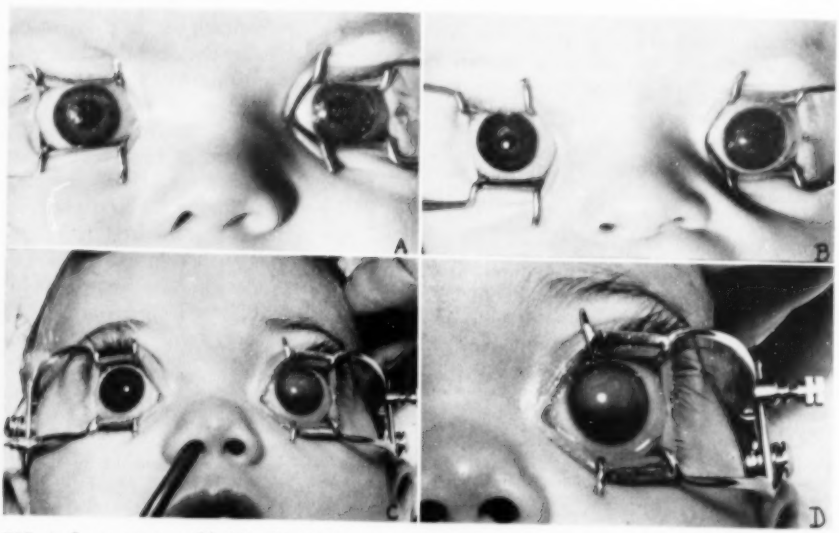

FIG. 1—Large corneas with (A) slight haze of left cornea; (B) moderate haze of left cornea; (C) marked opacity of left cornea. (D) Close-up of marked opacity of left cornea.

FIG. 2—Ruptures of Descemet's membrane.

that the disease is not present. An increasing corneal diameter is almost sure evidence of the disease; it is also of some value as a criterion for cure because with successful treatment, enlargement ceases. Corneal haze is usually an early finding and is first limited to the epithelium, although the stroma usually becomes involved in a short time. It may be almost imperceptible or of such severity that the cornea is almost opaque or porcelain-like. Early in the disease, corneal opacity is due to a disturbance of fluid metabolism associated with elevated intraocular pressure. As the process continues, cicatricial scarring takes place. Even this type of corneal opacity can clear remarkably, however, once the ocular tension is controlled. This can be true even of seemingly opaque corneas.

Early in the disease when the corneal haze is still limited to the epithelium, it may fluctuate rapidly with changes of ocular tension. The cornea may be clear one day when the tension is normal and very hazy the next day when the tension is elevated. Barkan[12] has demonstrated that when the haze is limited to the epithelium, the cornea can be cleared by removing the epithelium, as he advises in his present technique of goniotomy with contact lens.

Ruptures of Descemet's membrane invariably appear when there is a considerable degree of corneal enlargement. Their presence is an important confirmatory sign of glaucoma (fig. 2). Since they reflect severe damage by the glaucomatous process, it is quite obvious that under ideal circumstances the diagnosis should be made before they appear. The ruptures are usually horizontal and first involve the lower portion of the cornea. They may later increase in number and involve the upper portion of the cornea as well. They may be vertical or may even take the shape of an upright U. When multiple ruptures occur, sheets of Descemet's membrane may break away from the cornea between the ruptures and form a hyaline bridge across the apex of the cornea. As the cornea enlarges, Descemet's membrane at the margin of the rupture may pull away from the corneal stroma to form a hyaline shelf.

Glaucomatous excavation of the optic nerve is of very little value as a diagnostic sign in infantile glaucoma. It occurs very late in the disease and usually not during the first year after birth. Damage to the nerve is probably minimal because of the pliable elastic coat, which results in enlargement of the globe rather than distortion of the lamina cribrosa with destruction of the nerve fibers. When the view of the fundus is blurred due to corneal irregularity, Shaffer[38] suggests the use of a contact lens.

A most important corroborative sign, yet one which is often misleading, in the diagnosis of infantile glaucoma is the recorded increase in ocular tension. Several sources of error are apparent. The tension must be recorded with the child under deep surgical anesthesia, preferably with endotracheal intubation. The depth of the anesthesia should be such that the child is breathing freely and

completely relaxed. The eye should no longer be turned upward, which is indicative of light anesthesia, but instead should be in the primary position, which is characteristic of deep surgical anesthesia. The ocular tension may record as high as 40 mm. Hg Schiøtz even in a normal eye if the child is struggling or under light anesthesia. Such deceptive readings not infrequently lead to the mistaken diagnosis of infantile glaucoma and even to operative intervention. If any doubt exists and there are no other confirmatory signs of the disease, no harm is done by deferring surgery and repeating the examination in a few days. Barkan[14] has stated (and my experience has been likewise) that he sees a few patients each year for whom the diagnosis of infantile glaucoma has been made mistakenly because of false tension readings due to inadequate anesthesia. Likewise, too much reliance cannot be placed on a single finding of normal tension, because normal fluctuations of tension and such factors as dehydration associated with preparing infants for anesthesia may result in lowering the ocular tension. To re-emphasize, when any doubt exists as to the reliability of the tension reading, and certainly when the tension level conflicts with the remainder of the clinical picture, examination should be repeated on one or more occasions as indicated.

Another possible source of error in the recording of tension is the corneal curvature of large eyes, a fact mentioned by Barkan.[12] Although this might theoretically lead to a false low recording of ocular tension, in Barkan's and my own experience, if the tension remains under 27 or 28 mm. Hg Schiøtz, regardless of corneal curvature, the disease does not progress, and if the recordings are consistently 30 mm. Hg Schiøtz or over, the diagnosis is quite certain and deterioration follows.

Gonioscopy has been mentioned as a diagnostic aid, although in my own hands it is an unreliable one. Barkan,[12] Francois,[20] and Kluyskens[23] all mention its diagnostic value and describe persistent mesodermal tissue in the angle as characteristic of the disease. Much more work needs to be done on the subject before the method becomes dependable in diagnosis. It will probably never be of diagnostic value except in the hands of experts who have had a great deal of experience with this type of disease. The palisade-like arrangement of pigment at the insertion of the iris into the scleral spur, together with a filled-in apex of the angle of the anterior chamber, is thought to be the most characteristic part of the picture. However, many eyes with infantile glaucoma do not show these findings, and they are seen in some eyes that do not have infantile glaucoma. At the present time, evidence other than gonioscopic is necessary to make a diagnosis of infantile glaucoma. Kluyskens[23] mentioned iris atrophy as a possible diagnostic sign, but the appearance of the iris varies greatly in the normal eyes of children and as yet no truly characteristic picture of infantile glaucoma has evolved.

DIFFERENTIAL DIAGNOSIS

The enlarged cornea associated with infantile glaucoma must be differentiated from the large cornea of high myopia, megalocornea, Hurler's disease, and congenital idiopathic edema of the cornea.

The differentiation of infantile glaucoma from high myopia ordinarily gives very little difficulty. The corneal diameter is usually normal in high myopia; the eye presents none of the findings of infantile glaucoma just described and, of course, shows high myopia. One must be on guard that the two conditions do not occur together, because not infrequently high myopia is associated with infantile glaucoma.

Megalocornea occurs in an otherwise normal eye and is always present at birth. It is found predominantly in males, and it is bilateral and symmetrical. It is familial and almost always occurs in other members of the family. Visual function is good and the patient is symptomless.

The appearance of the eye when Hurler's disease is present may resemble that in cases of infantile glaucoma but the remainder of the clinical picture establishes the diagnosis.

Congenital idiopathic edema of the cornea, which is evidenced by diffuse epithelial edema, presents much more of a problem. These children often are photophobic; however, the corneal diameter is normal. Ruptures in Descemet's membrane do not develop and the ocular tension is normal. Repeated recording of ocular tension is usually necessary to exclude the diagnosis of infantile glaucoma.

Anderson[3] also includes anterior staphyloma, keratectasia, and keratoconus in the differential diagnosis but it is not necessary to mention the symptoms by which they are differentiated.

PROGNOSIS AND CLINICAL COURSE

The prognosis for infantile glaucoma has in the past been very dismal. Anderson[3] held out little hope for these patients. In the concluding paragraph of his book, he stated that the future of patients with this disease was dark and that, regardless of treatment, there was little hope of preserving sufficient sight to permit the earning of a livelihood. In his introductory paragraph, he warned that even though the tension may have been controlled, a series of degenerative changes had been set up which interfered with the nutrition of the ocular tissues to such an extent that ultimate blindness was almost inevitable. Kluyskens[23] stated that eventual blindness takes place in 20 per cent of the cases. He also stated that although the tension is controlled in 60 per cent, good function results in only 23 per cent.

The natural course of the disease, unless the tension is controlled, consists of slow progression of the classical signs previously described. The cornea and the eyeball continue to increase in size. The cornea varies from clear to semiopaque. The disease tends to stabilize somewhat when the child is from 12 to 14 years old, but gradual deterioration of the globe sets in. Thinning of the tissues becomes marked, particularly at the limbus, and rupture of the globe may finally occur from minor trauma. Such eyes are predisposed also to anterior chamber hemorrhage from trivial injuries. Retinal detachment and dislocation of the lens may occur. Loss of vision finally occurs because of rupture of the globe, corneal opacity, glaucomatous atrophy of the optic nerve and other degenerative changes.

Exceptionally, spontaneous regression may occur even after the disease has advanced to the point where all of the findings are well established. I have observed spontaneous regression in five eyes; in three cases it was unilateral. In all of these eyes, the classical picture of infantile glaucoma, with enlargement of the cornea and ruptures of Descemet's membrane, was seen. Repeated recording of ocular tension was normal following regression and the disease did not progress. Barkan[14] has also observed spontaneous regression, and Anderson[3] mentioned several instances.

TREATMENT

The treatment of infantile glaucoma is primarily surgical. The consensus of the members of the committee presenting this symposium is that deferment of surgery for medical therapy does nothing but deprive the patient of the better

result which might be anticipated from early surgery. It is the further opinion of the members of this committee that the operation of choice is goniotomy, an operation adapted to the treatment of infantile glaucoma as a result of the outstanding work of Barkan.[6-13] Because of Barkan's work, de Vincentiis[44] has suggested calling the operation de Vincentiis-Barkan operation. An increasing number of reports is appearing in the literature of the efficacy of the operation as described by Scheie,[35,37] Callahan,[16] McArevey,[26,27] McKinney,[28] Robertson,[32] Wilson,[54,55] Hertzberg,[22] Algan,[2] Bietti,[15] and Lister.[25] The technique will be discussed elsewhere in this symposium. Goniopuncture, recently described by Scheie,[36] also seems to offer some promise in the treatment of infantile glaucoma, either alone or when done in combination with goniotomy. Other operative procedures are best reserved for secondary operations where goniotomy or goniopuncture have failed to control the ocular tension. Neither goniotomy nor goniopuncture interferes with subsequent execution of other operative procedures such as iridencleisis, corneoscleral trephination and cyclodiathermy.

Results from procedures other than goniotomy or its outgrowth, goniopuncture, in the hands of this committee have been very discouraging. Such an opinion seems supported by statistics which have been quoted regarding prognosis for the disease. Zentmayer,[56] as a result of a survey in 1913, authoritatively pointed out the very poor outlook regardless of the type of surgical procedure. His review included corneoscleral trephination, iridencleisis, and cyclodialysis. In spite of such statistics and notwithstanding the opinion of this committee, reports continue to appear in the current literature advocating corneoscleral trephination, iridencleisis and other procedures. Sugar[39] states that of the operated cases, reports indicate that less than 10 per cent of patients have retained vision of 20/40 after two years. When comparisons are made of eyes which have and which have not been operated upon for congenital glaucoma, patients who have been operated upon are somewhat better off. Twenty-five per cent of operated patients are blind by the age of 12 years, as compared to 54 per cent of unoperated patients. By the age of 25 years, 45 per cent of operated patients are blind in comparison to 60 per cent of unoperated patients. Lister[25] tabulated the data of Anderson[3] and Kluyskens[23] (table I).

Pesme,[30] in a recent paper, advocated, as have many others, corneoscleral trephination. In the hands of this committee, the operation only rarely controls the tension and all too frequently is accompanied by vitreous loss, intraocular hemorrhage, subluxation of the lens, corneal edema and permanent opacity. Kluyskens,[23] Weekers and Weekers,[51] and Bailliart,[5] among others, advocate iridencleisis and report good results.

In recent years, cyclodiathermy has been offered as the operation of choice, particularly by European ophthalmologists. A recent technique, which involves applying electrodes for periods of 10 to 15 seconds 6 mm. or more back of the limbus, has rendered this method safer and of greater value in adult glaucoma, and this is probably true, also, of its use in the infantile type. Forbes,[19] Weekers and Weekers,[50] Alajmo and Simonelli,[1] Thomas and Algan,[41] Redslob,[31] and Calmette et al.,[17] all report good results from its use. The results experienced by members of this committee have not been as favorable, although the method has been used upon only a limited number of patients.

Goniopuncture

Goniopuncture was developed by Scheie[35,37] as an outgrowth of work with goniotomy. The operation consists of in-

TABLE I

QUOTED RESULTS OF OPERATIONS OTHER THAN GONIOTOMY

Operation	ANDERSON			KLUYSKENS			
	NUMBER	GOOD VISION PER CENT	6/60 VISION PER CENT	NUMBER	NORMAL TENSION PER CENT	GOOD VISION PER CENT	FAILURES PER CENT
Trephination	95	20	57	136	64	28	36
Iridencleisis	3	66	—	35	68	11	29
Iridectomy	27	0	72	—	—	—	—
Herbert's Sclerotomy	2	100	—	—	—	—	—
Anterior Sclerotomy	31	27	66	—	—	—	—
Cyclodialysis	13	8	92	—	—	—	—
Cyclodiathermy	—	—	—	44	77	30	30

troducing a goniotomy or goniopuncture knife into the anterior chamber, as in making a goniotomy, carrying the knife across the anterior chamber to the trabecular region of the corneoscleral wall, where an opening is made in the subconjunctival space. Although data accumulated are still insufficient to permit definite conclusions, it seems to offer some promise in the treatment of infantile glaucoma. When the outcome is successful, a permanent fistula is established which permits subconjunctival drainage and absorption of aqueous humor.

Fistulization is indicated by normalization of tension and diffuse edema of the conjunctiva overlying the puncture. The edema is so filmy and slight as to be almost imperceptible to the naked eye. An elevated polycystic bleb such as follows corneoscleral trephination does not develop. Gonioscopic examination reveals a gaping hole or cleft in the angle wall at the site of the puncture (fig. 3). Concrete evidence of filtration was found in three eyes with a juvenile type of pigmentary glaucoma, successfully operated upon by goniopuncture. During the two years following surgery, each had shown an increasing amount of pigment which apparently had washed into the subconjunctival tissues overlying the site of the puncture (fig. 3). The conjunctiva surmounting this area of the globe was edematous. The longest period of follow-up has been five years, but the ocular tension of several patients has been controlled successfully from two to five years. The procedure is of value only in patients under thirty years of age because in older patients a gaping cleft does not form in the corneoscleral wall.

Goniopuncture offers only a new method of performing an anterior sclerotomy, a procedure which has been done for many years. The first reference to anterior sclerotomy which I have found in the literature is that by de Wecker,[49] 1867, who suggested that such a procedure might be of value in glaucoma. In 1871, he described a technique for sclerotomy done by making a puncture and a counterpuncture just behind the limbus, enlarging each opening upward by a sawing movement of the Graefe knife.[48] A bridge of scleral tissue was left intact above these openings. Iris prolapse and staphyloma formation likewise occurred

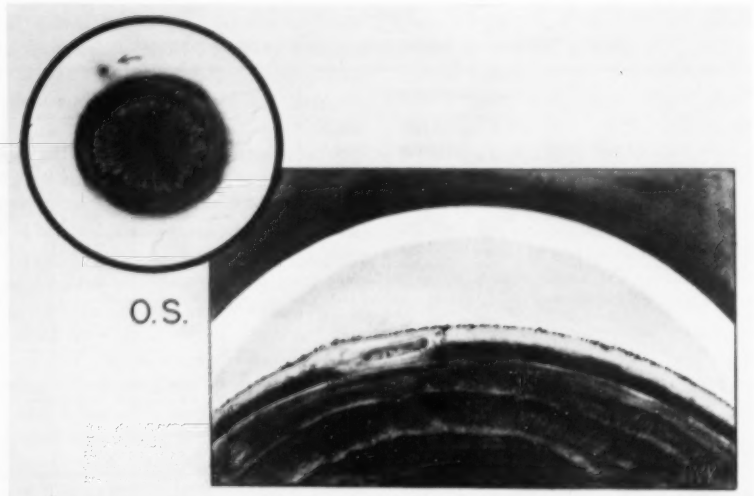

FIG. 3—Gaping cleft in the trabecular region at site of goniopuncture in an eye operated upon for juvenile glaucoma (pigmentary type). Inset shows pigment which has gradually accumulated in subconjunctival tissues as a result of aqueous flow.

at the site of the opening with this procedure. Following Tailor's report in 1891,[40] and that of de Vincentiis[45] in 1893, on the incision of the iris angle, de Wecker[46,47] modified his operation to combine the advantages of both procedures by incising the upper portion of the angle of the anterior chamber with the tip of the knife as it was withdrawn but not cutting entirely through the scleral bridge. He believed, however, that both operations were effective through incision of the sclera rather than debridement of the angle.

Rochon-Duvigneaud[34] employed a sclerotomy similar to de Wecker's, but he did not enlarge the openings nor did he incise the iris angle. His operation greatly resembled goniopuncture except that the conjunctiva was perforated at the site of the puncture and counterpuncture and two openings were made in the corneoscleral wall, whereas only one is necessary. One puncture is more likely to remain patent than two because of the greater amount of aqueous flow through a single one, which tends to fistulize the opening by the force of its flow. I have tried making the puncture from the external approach, patterning the technique after the Reese incision,[43] which has been practiced in years past in Boston, but the opening cannot be made as accurately as is possible when the puncture is made from within the anterior chamber. Another disadvantage of the sclerotomies of both Rochon-Duvigneaud and Reese is that if bleeding should occur following the puncture, blood cannot be washed from the anterior chamber, at the time the hemorrhage occurs, through an opening which is covered by conjunctiva. This can readily be done through the corneal opening at the site of entrance of the goniopuncture knife, and leaves the anterior chamber free of blood at the end of the operation. Barkan[12] recently has stated that he used a method similar to goniopuncture from 1936 to 1938, when he performed a limited counterpuncture of the sclera into the subconjunctival space in over twenty eyes in the belief that external fistuliza-

tion might be a factor in the action. None of his patients developed permanent fistulization; he therefore discontinued the practice.

I have, however, employed my method of goniopuncture in a sufficient number of eyes to know that it is of some value. Other reports of the successful use of goniopuncture have been submitted by Robertson,[33] Moore,[29] and Weisser.[52]

Since either goniotomy or goniopuncture can be effective as a primary procedure in controlling the ocular tension and apparently arresting infantile glaucoma, the question, which was raised by de Wecker and de Vincentiis, whether goniotomy works by incision of the corneoscleral wall or by debridement of the iris angle must be reopened. Valude and Duclos,[42] in reporting an anatomical study of 16 human infant eyes, pointed out how easily a fistula could be produced.

Goniopuncture seems to be particularly indicated in eyes where one or two previous goniotomies have failed. As pointed out by Anderson,[3] the canal of Schlemm may be absent congenitally or may become obliterated as enlargement of the eyeball takes place and the normal anatomy is distorted. In such eyes, goniotomy, which lowers ocular tension through re-establishing normal drainage of aqueous (at least theoretically), could hardly be expected to be successful. Goniopuncture, which actually produces a fistula, would seem a logical procedure, and it has been successful in controlling tension in at least some of these eyes. Because both procedures seem of value and since each, theoretically, has a different mode of action, I have been more and more frequently combining goniotomy and goniopuncture in the same operation. The sweeping incision of a goniotomy is made along the angle wall, and upon its completion, the knife is thrust through the corneoscleral wall to create a puncture, thus offering the advantages of both procedures. Although the number of patients so treated is not large, the incidence of success has seemed greater than when either operation was done alone.

Technique. The operation is done under general anesthesia in children. A preliminary oblique knife-needle puncture, as described by Chandler,[18] is made in the upper temporal quadrant to permit prompt restoration of the anterior chamber (fig. 4, A). Saline solution is injected through this opening after completion of the operation to aid in restoring the anterior chamber. This is desirable to help prevent the formation of an anterior synechia at the site of the puncture, which would close the fistula. The conjunctiva adjacent to the lower limbus is then ballooned outward away from the globe by the subconjunctival injection of saline solution (fig. 4, B). This maneuver prevents perforation of the conjunctiva by the knife when one is making the counterpuncture. A goniotomy knife is then introduced through clear cornea, 1 to 1.5 mm. within the limbus at approximately 9:30 o'clock in the right eye, or at 2:30 o'clock in the left eye, and directed nasally to the 6:00 o'clock meridian (fig. 4, C). The blade is introduced flat, on a plane parallel with the iris, and the tip is carried across the chamber until it reaches the trabecular region of the opposite angle. It is then thrust forward through the corneoscleral wall until the cutting end of the knife is entirely visible beneath the previously ballooned conjunctiva. It is then pulled back into the anterior chamber and withdrawn from the eye.

A Barkan goniotomy knife is better for the goniopuncture in infants than the especially designed goniopuncture knife which is used for adults. The goniotomy knife makes a smaller opening in the trabecular wall, which I believe is

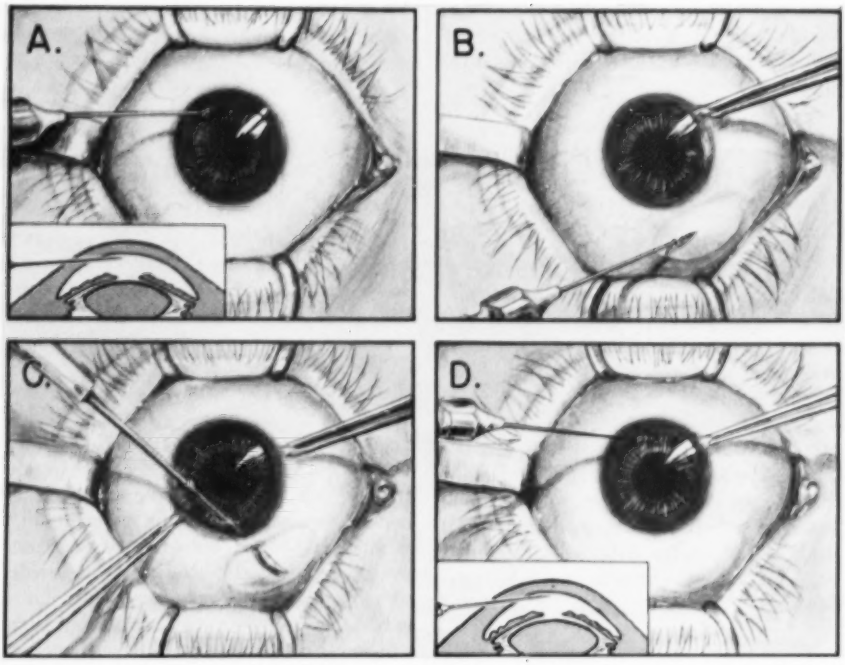

FIG. 4—(A) Oblique puncture with knife needle to prepare tract for injection of air or saline solution. (B) Raising of conjunctival wheal. (C) Goniopuncture knife blade carried across anterior chamber through corneoscleral wall of trabecular region. (D) Anterior chamber being filled with air or saline solution.

more desirable than the larger opening made by the blade of the bigger goniopuncture knife. A small opening is adequate in glaucomatous infant eyes because of the very thin corneoscleral wall. To keep the opening small, the knife should not be turned at right angles while the blade is in the subconjunctival space, as is advised in adults. Since the knife, therefore, is drawn back into the anterior chamber in the same plane in which it entered the subconjunctival space, the blunt heel of the goniotomy knife offers no disadvantage.

The puncture through the trabecula should be only a puncture, and temptation to enlarge it as the knife traverses the corneoscleral wall must be resisted. A larger opening leads to incarceration of the iris, with obstruction of the opening, and even if this does not happen, permanent fistulization develops less frequently, possibly because the force of outflow of aqueous diminishes with large or multiple punctures. Occasionally, bleeding occurs at the time of the puncture but causes no difficulty if the blood is washed from the anterior chamber at once. This is easily accomplished by inserting the tip of a blunted 27-gauge needle into the opening at the site of entrance of the goniopuncture knife into the anterior chamber.

The last step in the operation involves re-establishing the anterior chamber. A hypodermic needle is introduced into the knife-needle tract, as described by Chandler,[18] without entering the anterior chamber. Saline or air, then injected (fig. 4, D), is trapped in the anterior

chamber by the inner aspect of the knife-needle incision, which as a result of being long and very beveled behaves in a traplike manner. Difficulty not infrequently is encountered in filling the anterior chamber following goniopuncture for infantile glaucoma because of the thin corneoscleral wall which allows free communication between the anterior chamber and the subconjunctival space. Air or saline injected into the chamber may escape into the subconjunctival space and compress the globe, thus further preventing formation of the chamber. If this is seen to occur, further attempts should not be made to restore the chamber. Both eyes should be covered for twenty-four hours. Cortisone or hydrocortisone drops can be instilled for a few days postoperatively.

DISCUSSION

The importance of early diagnosis of infantile glaucoma is stressed because of the favorable response to surgery done before the eye is badly damaged by the disease. The diagnosis is readily made when all of the classical signs of the disease are present. However, it can be as difficult and obscure as the recognition of chronic simple glaucoma of the adult early in the disease. Photophobia, especially during the first year of life, whether or not the corneas are clear, should lead the physician to suspect the presence of infantile glaucoma.

Examination and recording of tension under deep surgical general anesthesia, preferably with endotracheal intubation, should be done in all suspected cases. The pitfalls of recording tension under anesthesia have been mentioned briefly. The two greatest sources of error are insufficient anesthesia to cause complete relaxation, which results in deceptively high readings, and dehydration from withholding fluids during preparation for anesthesia, with low readings as the result. A single recording of tension should not be trusted if it conflicts with the clinical picture, but it should be repeated on one or more other occasions.

The clinical course in the past has been discouraging, as pointed out by Anderson and others. Except for an occasional spontaneous remission, all patients become blind on a medical regimen. Conventional, long established, surgical methods have met with mediocre success. It is the opinion of the committee presenting this symposium that goniotomy is the treatment of choice, either alone or combined with goniopuncture. The results of various surgical methods are given elsewhere in the symposium. It is the belief of this committee that operations such as corneoscleral trephination, iridencleisis, cyclodialysis and cyclodiathermy should be reserved for secondary operations after goniotomy or goniopuncture have failed. A trial of medical treatment only wastes valuable time and subjects the eye to damage by the disease, which interferes with the success of later operations.

SUMMARY

1. Early diagnosis is emphasized.
2. Photophobia in a child under one year of age should immediately suggest the possible diagnosis of infantile glaucoma.
3. Deep surgical general anesthesia, preferably with endotracheal intubation, should be used when the tension is recorded.
4. A trial of medical therapy only wastes valuable time because failure almost invariably results and the eye develops changes which interfere with the success of surgery.
5. Goniotomy or goniopuncture, alone or combined, is the surgical methods of choice.

6. Corneoscleral trephination, iridencleisis, cyclodialysis and cyclodiathermy should be reserved for secondary procedures.

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GONIOTOMY

OTTO BARKAN, M.D.
SAN FRANCISCO, CALIFORNIA

FIG. 1—Microgonioscopic view of angle in a case of infantile glaucoma (age 7 weeks). The iris is shown proceeding horizontally to its anterior insertion into the trabecula. The exposed area of trabecula is somewhat opaque, indicating diminished permeability. The incision should be made close to the line of Schwalbe as indicated in figures 2 and 3.

THE rationale of goniotomy is based on the pathologic anatomy and pathogenesis of congenital or infantile glaucoma. The pathogenesis, which has been evolved from microscopic findings and on the basis of gonioscopy, has been set forth in the first section of this symposium.[1-5,12-14,16,17,21]

De Vincentiis, in 1892, was the first to introduce a surgical approach to the angle across the anterior chamber.[23,24] After the blade was guided across the chamber, it disappeared from view behind the limbus, a blind or semiblind operation. The procedure fell into disuse because of occasional severe hemorrhage and other complications. It was later combined with sclerotomy in the attempt to obtain external fistulization. The results were indifferent.

In 1934 the writer devised a method of operating in the angle with visualization by means of a contact glass.

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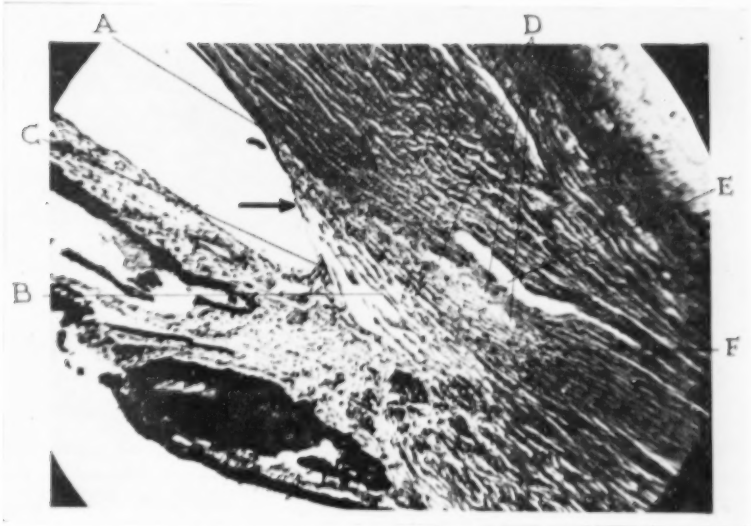

FIG. 2—Section (taken from Seefelder) of the angle region in a seven-day-old infant with congenital glaucoma. The arrow indicates the point where the incision should be made. (A) the ring of Schwalbe; (B) the anomalously differentiated trabecula; (C) at the anterior insertion of the iris is the hook formation described by Seefelder. This corresponds to the tentlike folds and the seamlike attachment seen in the gonioscopic picture (fig. 1). The oblique plane of the iris is considered to be an artefact since in vivo, as seen gonioscopically, the plane of the iris is horizontal. (D) the canal of Schlemm. (E) a collector channel. (F) the scleral spur.

Because of the rarity of congenital or infantile glaucoma, it has taken years to establish the rationale and to perfect the technique of operation. The results of postoperative gonioscopic studies have been helpful in further evolving the concepts of pathogenesis.[5,6]

The objective of goniotomy is to remove the obstruction which is causing the retention of aqueous and to give access for aqueous to the Schlemm's canal system, thus restoring and maintaining the physiological direction of outflow.

The anomalous trabecula covering Schlemm's canal can be seen gonioscopically (fig. 1). The picture corresponds, in my experience, to the findings in anatomic sections in which the iris in many cases inserts anteriorly into the trabecula (fig. 2, C). The incision of the anomalous trabecula should be superficial and close to the line of Schwalbe (figs. 2 and 3) in order to avoid injury to the underlying outflow mechanism and to the root of the iris, with consequent hemorrhage, scars and adhesions. There has been no evidence of external fistulization or of a cleft communicating with the suprachoroidal space in any case in my series. The iris and scalloped pigment border which appear to have been pulled or pinned up in folds or tents toward the ring of Schwalbe previously, drop backwards. The incision exposes the Schlemm's canal drainage system to aqueous (fig. 3).

In the normal infant, Schlemm's canal, which frequently fills with blood on application of the contact glass, is covered by an almost transparent layer of tissue, the normally permeable trabecula (fig. 4). In congenital or in-

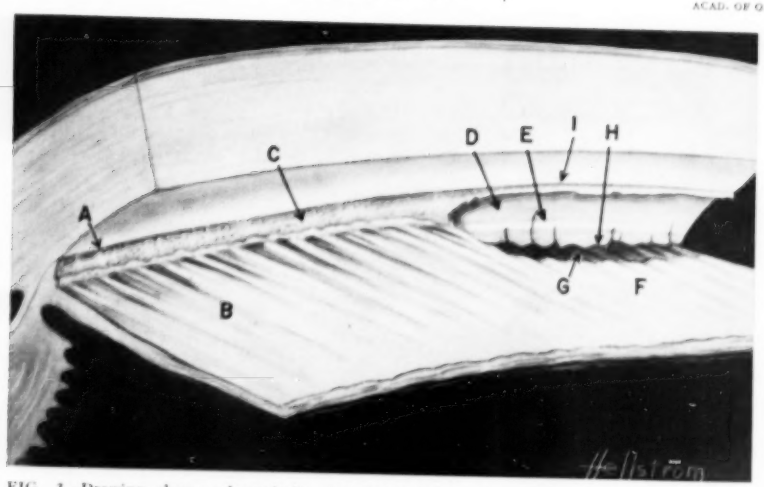

FIG. 3—Drawing shows schematically the postoperative microgonioscopic appearance at the point of transition between the area on the right, in which the angle has been operated upon, and the neighboring untouched region. (A) Ring of Schwalbe. (B) The plane of the iris is horizontal. It inserts by means of a wavy line into a semitranslucent cellophane-like shagreened surface. This is the inner surface of the anomalously differentiated trabecula which crosses the angle in anatomic sections, as shown in figure 2. (C) The cellophane-like surface inserts into the ring of Schwalbe in an almost vertical plane. The width of this surface, or membrane, shows many variations according to the degree of anterior insertion of the iris. (D) Angle wall exposed. (E) Scleral spur. (F) Iris stroma, released and recessed from its attachment to the ring of Schwalbe. (G) The segments of pigment epithelium lie in a more posterior plane. (H) Insertion of iris to angle wall. (I) Note ring of Schwalbe curving slightly upward in the region in which the attachment of the iris has been separated from it. (The cross section on the left of the drawing is a schematic representation of anatomic sections.)

fantile glaucoma, Schlemm's canal, when filled with blood by jugular compression, as suggested by Scheie, is veiled by overlying tissue, the anomalous trabecula, or it may even be entirely hidden by the anterior insertion of the iris (fig. 5). In most surgically normalized eyes, the filling of Schlemm's canal with blood demonstrates its presence and shows its position and the effect of the incision in exposing Schlemm's canal to aqueous (fig. 6). When the overlying tissue (the anomalous trabecula) is effectively incised, it separates and the pressure is normalized. In many cases the root of the iris drops backwards. It is evident from the illustrations how superficial an effective incision must be if injury to the underlying drainage system and scarring of the angle wall are to be avoided. This emphasizes the importance of operating under visualization. Experience has shown that there should be no scraping, no debridement, no deep incision nor splitting of the sclera.

TECHNIQUE

In the course of years goniotomy under visual control has evolved into a simple, though precise, procedure for one who is adept at gonioscopy and who has familiarized himself thoroughly with the details of its technique. It is a question of minutiae upon which depend not, as in older adults, a few years of life but a whole lifetime of vision.

Preoperative and postoperative gonioscopic examinations are made in all cases with the binocular microscope mounted on an adjustable and movable floor

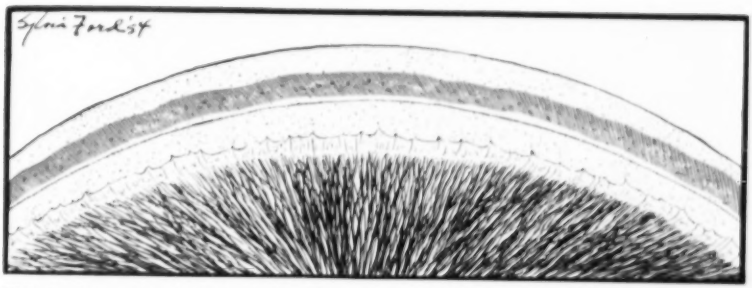

FIG. 4—Gonioscopic view of normal angle in an infant, showing transparency of tissue overlying the blood-filled Schlemm's canal.

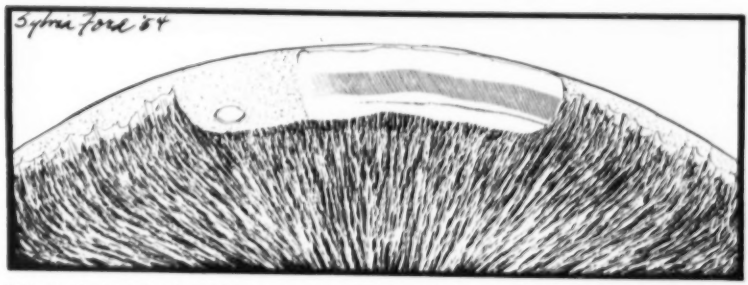

FIG. 5—Gonioscopic view of angle in a case of infantile glaucoma (age 4 months) after operation by goniotomy. In the unoperated area the anterior insertion of the iris hides Schlemm's canal from view. After incision near the line of Schwalbe, the iris has dropped back exposing Schlemm's canal to aqueous. To the left of the incised area some of the veiling tissue (anomalous trabecula) is still present.

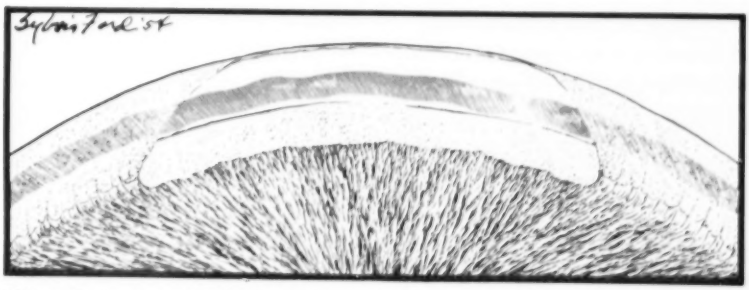

FIG. 6—Gonioscopic view of angle in a case of infantile glaucoma (age of child, 3 months) after operation by goniotomy. In this case the insertion of the iris is normal. The trabecula veiling Schlemm's canal is less transparent than normal. Its incision has exposed Schlemm's canal to aqueous.

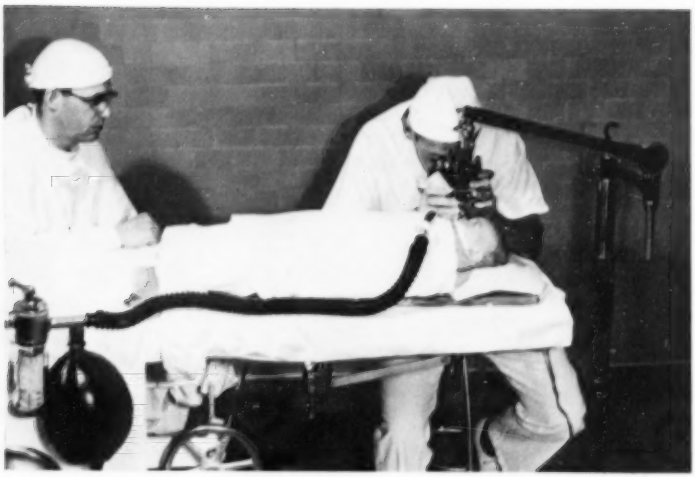

FIG. 7—Examination of infant under intratracheal anesthesia with microgonioscope and focal illuminator.

stand, using a focal illuminator[7] and with the patient under intratracheal ether anesthesia (fig. 7).

The technical difficulties encountered in the early stages of development of the procedure have long since been overcome. To overcome these initial difficulties, modifications of the technique, such as injection of air or of saline solution,[15,22] were devised, but I find they merely complicate the present simplified technique.

GONIOTOMY WITH VISUALIZATION

At the present time the procedure may be summarized as follows:

Preoperatively, a pediatric examination, including a roentgenogram of the chest to determine the state of the thymus, is performed. Maximal miosis is obtained by the instillation of drops of prostigmine (5 per cent) three times at half-hour intervals before operation. Ether anesthesia is administered by the intratracheal method. This assures a generous supply of oxygen as well as complete relaxation of the patient, which is important if the true measurement of the intraocular pressure is to be obtained. Before the patient is draped, the pressure in the eye is measured with a tonometer, the base of which has been sterilized. The corneal diameters are measured with a caliper, and the condition of the corneas and size of the pupils are noted.

The infant is placed on a circumcision board to facilitate adjustment of its position. The head rests on a sponge rubber ring. The face is prepared and the eye irrigated in the usual manner. A face mask of dark color is used to prevent glare.

A canthotomy may be performed after the injection of a few minums of adrenalin (1:1000) into the outer canthus. The speculum, which has been passed through two holes in a dark rubber dam six inches square, is inserted and the rubber dam trimmed.

The surgeon operates in the standing position, almost upright, and facing the patient at the level of the shoulders. The

eye should be 47 inches above the floor for a surgeon of average height. The cornea is kept moist with physiologic saline solution applied with a glass rod. It is advisable for a presbyopic surgeon to wear a correction for distance of seven inches.

For illumination, a nurse, who stands on a stool, holds an air-cooled plastic hammer lamp in contact with the right temple of the surgeon while looking down the top of the lamp so that she may have the same view of the angle as the surgeon.

The assistant fixates the eye with two Gifford forceps, sharply pointed and with spring lock, applied to the insertion of the tendons in the vertical meridian.

If corneal cloudiness prevents or disturbs visibility under the glass, a preliminary abrasion or denuding of the clouded epithelium with a keratome is performed. This should be complete and should be repeated until maximum visibility is obtained.

Before application of the contact glass, the assistant rotates the head to the contralateral side, with the temporal limbus of the eye to be operated upon pointing upwards. With the head in this position, the surgeon is operating downhill. This prevents the ingress of air under the glass. The surgical contact glass (medium size) is applied in the usual manner by injecting physiological saline solution between it and the cornea through a curved gold cannula and a 1.5 cc. Luer syringe. The surgeon controls the glass with the index finger of his left hand. Two indentations on top of the glass prevent it from slipping under the finger.

The surgeon places the glass on the nasal part of the cornea in order to expose a temporal sector of the cornea, two to three millimeters wide, where the puncture is to be made. The eye is abducted slightly to allow ample room in which to push the glass nasally (canthotomy facilitates this) and thus keep the temporal crescent of cornea exposed.

Only one eye is operated on at one session. The puncture is made in the right eye at the ten o'clock position and in the left eye at the four o'clock position, 1 mm. axial to the corneoscleral border and a little obliquely so that the corneal wound is like a valve or trapdoor. It is between 1 and 2 mm. long. It must not be too oblique lest it impede the passage of the tapered shaft of the knife as it traverses the chamber. The anterior chamber is never lost if a correctly tapered knife[8] is used and if the knife is not retracted during the operation. The importance of the rotated position of the head and of the correct taper of the knife cannot be overemphasized.

After crossing the anterior chamber, the point of the blade is inserted into the abnormal trabecula posterior to the ring of Schwalbe. The depth of the incision should not be more than one fourth of a millimeter; that is, the blade, which is 1 mm. long, should not disappear in the tissue for more than one quarter of its length. It is moved counterclockwise in this plane, a distance of several millimeters, or one third or more of the circumference. Experience has shown that the more superficial the blade remains, the better are the results.

An incision of the scleral wall is avoided. During the excursion of the blade, the shaft is rotated slightly around its axis in a clockwise direction to prevent it from moving posteriorly out of the intended plane into the region of the root of the iris. The iris retracts and falls backward, leaving a light colored wake behind the blade. The incision is under complete visual control (fig. 8). When it has been completed, the knife is removed quickly, with pressure exerted against the back of the blade in order to

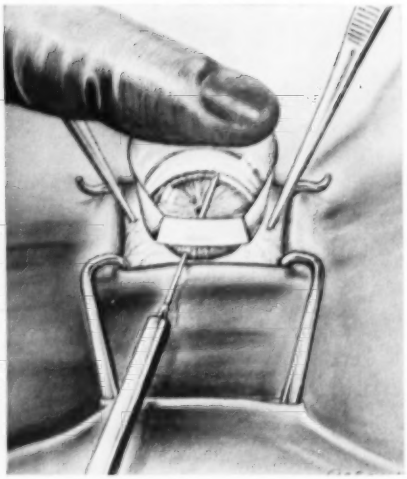

FIG. 8.—Goniotomy of the angle under the glass. The root of the iris retracts behind the blade, leaving in its wake the wall of the angle.

prevent enlargement of the puncture wound of the cornea. Contact glass and fixation forceps are removed. After a few seconds, a slight capillary oozing of blood, venous in color, may appear in the anterior chamber along the line of incision; this is absorbed within a few hours.

The head is rotated so that it rests on the operated side. With a curved gold cannula and a 1.5 cc. Luer syringe, air is injected through the puncture wound into the anterior chamber, inflating it from one-half to two-thirds of its normal size. If the head is rotated so that the bubble rises into the operative site, the raw surfaces of the angle are kept apart and the air is locked in the chamber. If the miotic pupil is completely covered by air, some degree of pupillary block may be observed to produce a transitory bombé of the iris in its dependent portion.[9,20] The pupillary block can be relieved, if desired, by removal of air, or by shifting the air bubble from the pupil by means of external pressure on the cornea with the tip of a glass rod. I have never seen an irreversible iris bombé or a glaucoma due to air develop following goniotomy in infants.

The speculum is removed and the canthotomy closed with catgut. One drop of di-isopropyl fluorophosphate (DFP) and sulfacetimide ointment (10 per cent) are inserted into the conjunctival sac.

Binocular pads are applied, the pad over the operated eye being covered with a metal shield. For the first 24 hours the child is kept lying on the side corresponding to the operated eye, so that the blood, if any, will settle on the opposite side of the chamber and the air will keep the raw surfaces of the operated region apart.

In bilateral glaucoma, prostigmine (5 per cent) and one drop of DFP, 0.1 per cent, are instilled in the unoperated eye after 24 hours, and in the operated eye after 48 hours. Drops of postigmine (5 per cent) are continued four times a day in both eyes during convalescence.

If subsequent operations are necessary, they can be performed on other quadrants of the angle, with the assistant rotating the globe around its axis. The operation may be repeated within two weeks. In several cases in which the nasal angle was scarred as the result of previous goniotomies or cyclodialyses, a good result was obtained by operating across the nose on the untouched temporal angle.

Abrasion or total denuding of the clouded corneal epithelium with a keratome made it possible to operate under gonioscopic control with the glass in many cases in which this otherwise would have been impossible. In the last 100 eyes operated upon, 94 were operated upon under gonioscopic control with the glass. In four of the six eyes in which the operation was performed without the glass because of corneal

cloudiness, the reduction of pressure was only partial. However, the cornea cleared sufficiently so that the next operation could be performed with the glass.

Ruptures of Descemet's membrane sometimes make the operation under gonioscopic control a little more difficult because of the aberration which they cause, but they can always be circumvented by adjustments of position and changes in the line of observation.

The point of the blade of the knife should be precisely localized in depth in order to assure a superficial incision of the thin layer of aberrant tissue. Since in most infants the eyes are blue and the stroma transparent, the serrated edge of the pigment segments of the epithelial layer at the root of the iris is a prominent feature of gonioscopy and makes an excellent landmark by which to judge where the point of the blade is to be placed, that is, close to the line of Schwalbe.

In the presence of a dark iris, the localization of the point in depth is more difficult because of lack of light in the angle and because the pigment serrations which provide a striking landmark in blue eyes are absent. In dark eyes it is therefore advantageous to use a 2-power loupe, which makes it possible to see details of the insertion of the dark iris. This outweighs the sacrifice of depth of focus which is the result of the increased magnification.

GONIOTOMY WITHOUT THE GLASS

In the presence of marked corneal parenchymatous scarring which cannot be relieved by denuding the clouded epithelium, the periphery of the cornea is usually clear enough for incision of the angle to be tried without aid of the contact glass by a technique which is based on that of de Vincentiis.[24] For goniotomy without the glass, the patient's head is placed in a horizontal position, with the height of the eye such that the operator looks vertically down on it. Good results can frequently be obtained in this manner. The puncture in the cornea is made obliquely in order to encourage retention and reformation of the anterior chamber. The blade of the knife crosses the pupil and disappears behind the limbus on the opposite side in the plane just anterior to that of the iris, its tip being visible through the sclera from the outside. Seen through the cornea, the knife appears to be 0.5 mm. more anterior than it actually is. It is better to err on the conservative side by maintaining a more anterior plane, even at the risk of missing the objective. Air is injected at the completion of the operation to keep the raw surfaces apart.

Hazards are associated with the semiblind or blind technique. Accurate removal or incision of the thin superficial layer of obstructive tissue cannot be accomplished with the precision and consistency which is made possible by operating with visualization, that is, under magnified gonioscopic control. There is the fundamental difference between the procedure of operating blindly and that by direct vision, namely, that of seeing or not seeing.

INDICATIONS

Goniotomy is indicated in all cases of congenital or infantile glaucoma in which increased pressure is the result of obstruction of the angle by a characteristic developmental anomaly, provided that Schlemm's canal has not been obliterated by prolonged distention of the globe. The characteristic anomaly in the angle is present in almost all cases of infantile glaucoma.

It is suggested that goniotomy be performed as the first operation and that if this procedure fails to normalize the

tension, it be repeated as much as four times on adjacent quadrants before resorting to other surgical procedures.

The earlier goniotomy is performed after the onset of symptoms, the more effective it will be anatomically in normalizing pressure and functionally in maintaining or restoring vision. Early diagnosis and operation are of utmost importance.

Goniotomy may also be effective and satisfactory visual results obtained in cases in which the onset of symptoms has been delayed or after the lapse of several years following the onset of increased pressure provided that (1) the glaucoma has not been congestive, (2) the cornea has remained clear, (3) the optic nerve has undergone little damage, and (4) the globe is not excessively enlarged. Excellent results have been achieved in a number of such cases in which these conditions were present in children 4 and 6 years of age with corneal diameters of 14 mm. Goniotomy has also proved successful in one case of glaucoma associated with aniridia[10] and in two out of three cases of glaucoma associated with naevus flammeus or oculocutaneous angiomatosis.[5,11]

CONTRAINDICATIONS

Goniotomy is usually contraindicated for older children whose corneas have a diameter of 15 mm. or more. In these distended "buphthalmic" eyes the danger of hemorrhage is increased by dilated vessels. Schlemm's canal may have been obliterated as a result of the distention. Dislocation of the lens or retinal detachment may result from the sudden decompression which follows perforating surgical procedures performed in this stage.

Goniotomy may also be ineffective in those severe cases in which the cornea is white at the time of birth. Probably the clinical severity of these cases indicates a correspondingly severe developmental anomaly which may be associated with absence of Schlemm's canal. Nevertheless, some of these cases have been normalized by goniotomy. However, the prognosis is not so good for normalization in them as it is in those in which the congestive onset is delayed until the third month or later.

SAFETY OF GONIOTOMY

Goniotomy, when performed by the technique described, does no damage. In the last 150 eyes operated upon since the development of the present technique, no complication has occurred. Sympathetic ophthalmia has never occurred in the total of 212 eyes.

If goniotomy is unsuccessful, other operations can be performed subsequently without disadvantage. The attempt may be made to obtain external fistulization by anterior or internal sclerotomy,[18,25,26] which is still used occasionally in Europe, or by Scheie's goniopuncture,[19] or a cyclodiathermy may be performed before resorting to the more radical operation of iridencleisis. In my personal experience with twelve eyes recently operated upon by goniopuncture, it has been ineffective in producing permanent results. Early in my series, a limited counterpuncture or sclerotomy into the subconjunctiva was added to the goniotomy operation in twenty eyes, none of which obtained permanent fistulization. The results of cyclodiathermy have not been encouraging. To be certain that a result following a fistulizing operation or cyclodiathermy is permanent, a follow-up period of at least eight months is necessary. Serious complications may follow both cyclodiathermy and iridencleisis. Sympathetic ophthal-

mia after both has been reported in the literature. I have observed it following iridencleisis in one case which was seen in consultation.

ADVANTAGES AND DISADVANTAGES

The great advantages of goniotomy when performed with visualization are its safety and absence of mutilation or disfigurement. It creates no pathologic complication while restoring the physiologic direction of outflow.

The disadvantage of goniotomy has been the frequent need of repeated operations. With the present technique, this disadvantage has been largely overcome.

VISUAL RESULTS

Increased intraocular pressure in infants usually causes corneal cloudiness. If allowed to persist, corneal cloudiness produces permanent irregular astigmatism and scarring of the parenchyma of the cornea. The corneal disturbance interferes with the development of central acuity and amblyopia results.

At this tender age a considerable degree of amblyopia may occur within one or two weeks as shown, for instance, by the development of nystagmoid movements during the course of two weeks and their disappearance when the cornea was cleared by goniotomy.

In congenital or infantile glaucoma the optic disc and fundus often appear blurred to direct ophthalmoscopic examination even though the cornea appears clear to casual inspection. If in these cases ophthalmoscopy is performed through the contact glass, an exquisitely detailed picture of disc and fundus is obtained.[5] The blurred image has been cleared by the contact glass. The explanation appears to be that irregular astigmatism, which is the residue of previous corneal cloudiness, causes the blurring of the image, and that this astigmatism is eliminated by the contact glass. By the same token, the reduced visual acuity in these cases is presumably an amblyopia, acquired during the period of early cloudiness and its resultant irregular astigmatism.

In the ultimate visual acuity, injury to the optic nerve is a much less important factor than disturbance of the cornea. Of course, in cases with unduly delayed normalization of tension it must be anticipated and prevented. Ultimate visual acuity depends largely upon how soon after onset the pressure and cloudiness are relieved. In the early stages the corneal changes are reversible. This emphasizes the urgent need of early diagnosis and prompt operation. The use of diamox should not be permitted to delay operation. At the present time it is useful between operations and when delay is unavoidable. Further experience may extend its uses.

CONCLUSION

Goniotomy with the glass under gonioscopic control should be performed as soon after onset of the increased pressure as possible. In case of cloudy cornea, removal of the cloudy epithelium permits operation with the glass, with visualization in over 90 per cent of cases. The incision of the anomalous tissue covering the Schlemm's canal system should be superficial. The operation with visualization is without hazard for a surgeon who has thoroughly familiarized himself with it and gonioscopy.

Goniotomy restores the physiologic direction of outflow without creating a pathologic condition.

An urgent plea is made for early diagnosis and early goniotomy with visualization of the angle.

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END RESULTS OF TREATMENT

JOSEPH S. HAAS, M.D.
CHICAGO, ILLINOIS

THE outcome of the treatment of 329 eyes of 202 patients with the infantile form of congenital glaucoma is discussed.

Infantile glaucoma, although rare, has been responsible for a disproportionate amount of frustration and concern to the ophthalmologist not only because of the ocular distress of these pathetic infants but also because of our gross inadequacy to combat its destructive characteristics. The motivation in undertaking this analysis has been the desire to alleviate this situation by emphasizing the urgency of immediate and proper therapy.

The material for this paper has been contributed largely by Dr. Otto Barkan, who has made available the extensive material from his private practice. Dr. Harold Scheie and Dr. Robert Shaffer have also contributed the histories of patients from their private practices. The remainder of the material is composed of cases from the glaucoma clinic of the Illinois Eye and Ear Infirmary, coupled with those of the author.

All of the eyes included have been afflicted with infantile glaucoma, i.e., the elevation of the intraocular pressure has occurred during the period when the ocular coat was still distensible. They have all demonstrated an elevated tension under deep anesthesia and most of them have had one or more of the following conditions: increased corneal diameter, corneal clouding, rupture of Descemet's membrane, or occasionally, glaucomatous optic atrophy. Those eyes which contained other congenital abnormalities, such as aniridia, neurofibromatosis, and nevus flammeus, were not included in this study.

The history of each eye was reviewed and an evaluation of the response to individual therapeutic measures made. It was difficult to select an accurate index of the control of these patients.

TABLE I

SOURCE OF MATERIAL INCLUDED IN THIS ANALYSIS

	PATIENTS	EYES
Barkan	130	210
Scheie	45	73
Shaffer	9	16
Haas	18	30

Vision was felt to be an unacceptable guide, for an evaluation of the preoperative vision cannot be made, and for most of the patients it has been impossible to obtain a measure of postoperative vision. It should also be emphasized that the postoperative vision is a result of the lack of treatment as often as of the treatment.

Inasmuch as none of the panel has observed progress of the physical signs of infantile glaucoma with the tension normalized, it was felt that those eyes

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which without miotics revealed a normal tension (in this situation below 28 mm. Hg Schiøtz) three months after surgery and in which no progress of the disease could be perceived could be considered normalized. Additional justification for this concept is provided by the fact that Barkan[3] has seen only ten cases of recurrence out of 196 normalized eyes in the past seventeen years.

RESULTS OF MEDICAL TREATMENT

All of the 329 eyes in this series were subjected to miotic therapy. The miotics used included pilocarpine, carcholin, eserine, prostigmine, and di-isopropyl fluorophosphate. The tensions recorded at the initial examination under general anesthesia are shown in figure 1. Initially ten eyes were normalized by miotics, but none could be maintained at this level or safely controlled.

The panel, therefore, agrees with Arnold, Lodata, DeLapersonne, LaGrange and the many others quoted by Anderson,[1] who have pointed out the danger of miotics when they lead to the postponement of the operation. Rather, the panel would view miotics as an aid to operative measures in restoring and maintaining the normal tension. When miotics lower the tension, they have a very definite preoperative value; and when the tension persists after operation, their use is certainly indicated between operations.

Acetazoleamide (Diamox)

In recent months the possibility of controlling the tension by inhibiting the formation of aqueous with diamox, while hoping that further maturation of the eye will allow the so-called immature angle to proceed to its normal state, has been postulated by Becker.[4] Becker reports a total of three infants in St. Louis and two in Baltimore who are under maintained therapy with diamox. The tension in the eyes of these children is

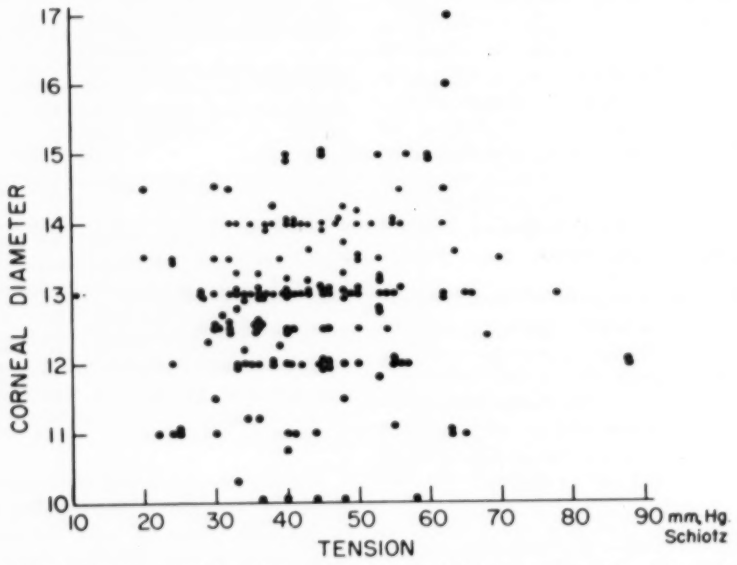

FIG. 1—Tensions recorded at the initial examination with 181 eyes with congenital glaucoma, following therapeutic use of miotics.

under excellent control, but too little time has passed to evaluate what is happening to the disease process, whether the mechanism responsible for infantile glaucoma is being altered, or whether the infants can safely tolerate maintained diamox therapy.

SURGICAL TREATMENT

Goniotomy

Two hundred and fifty-three eyes were operated upon by goniotomy. Of these the tension was normalized in 114 by the first operation. Goniotomy was repeated on 120 eyes, and 52 were normalized. When the operation was done on 66 eyes for the third time, 10 were normalized. A similar number responded to the fourth goniotomy, when 10 of 55 eyes were controlled. Eight more responded to the fifth goniotomy, bringing the total number of normalized eyes to 194 or 77 per cent of the original 253.

An attempt to explain the reason for failure of the operation produced several interesting facts. It was apparent that if the onset of infantile glaucoma occurred at birth or during the first two months after birth, it was more resistant to treatment. Of 69 eyes in which the onset of glaucoma occurred before the third month after birth, the total percentage of cure was only 55. Of even greater significance is the fact that it required 149 goniotomies to normalize tension in the eyes of these 38 patients (table III).

When the onset occurred during the second two months there was a startling difference in prognosis. Of these cases, the total normalized was 97 per cent, whereas the incidence of success per goniotomy had risen to 72 per cent.

It has been difficult to account for the difference in prognosis for those cases occurring during the first two months after birth. It was found in our study

TABLE II

NUMBER OF EYES IN WHICH TENSION WAS NORMALIZED BY ONE OR MULTIPLE GONIOTOMIES (253 CASES)

GONIOTOMIES REQUIRED	EYES NORMALIZED
1	114
2	52
3	10
4	10
5	8
Total	194 or 77%

TABLE III

PROGNOSIS OF CONGENITAL (INFANTILE) GLAUCOMA

AGE AT ONSET	NUMBER OF EYES	NUMBER NORMALIZED	PER CENT NORMALIZED	NUMBER OF GONIOTOMIES TO NORMALIZE	PER CENT SUCCESS PER GONIOTOMY
From birth through two months	69	38	55	149	25
From two months through four months	38	36	97	50	72
From two months through seven months	58	53	91	77	69

that none of the goniotomies succeeded in eyes with a corneal diameter greater than 14 millimeters. An examination of the diameters of the corneas of infants in the youngest age group revealed that none exceeded this measurement. The possibility of the cornea being so small as to increase the difficulties of operation could also be discarded (fig. 2). It seemed probable that the difference in prognosis could best be explained by a difference in the severity of the disease. This assumption gained support from the observation that both the corneal clouding (fig. 3) and the initial tension (fig. 4) were proportionately higher during the first two months.

It is unfortunate that the visual acuity of these patients is so difficult to analyze. As has been pointed out, it is impossible to obtain a measure of the initial visual acuity. Furthermore, although much time has passed since the first goniotomy was performed, the majority of the children in this study are still too young for us to make a satisfactory determination of their final vision. A truly significant analysis of the resultant visual acuity must await the passage of many years.

A dramatic improvement in the visual prognosis is suggested by those cases in which study of the visual acuity and adequate follow-up have been possible (table IV).

Goniopuncture

Seventeen patients in this series have had primary goniopunctures, ten of which resulted in normalization of the tension. These ten cases have been observed for varying periods: one for four years; one for three and one half years; one for two and one half years; two for two years; two for one year; and three for five months, without recurrence. When goniopuncture was repeated in two cases, it was successful in one.

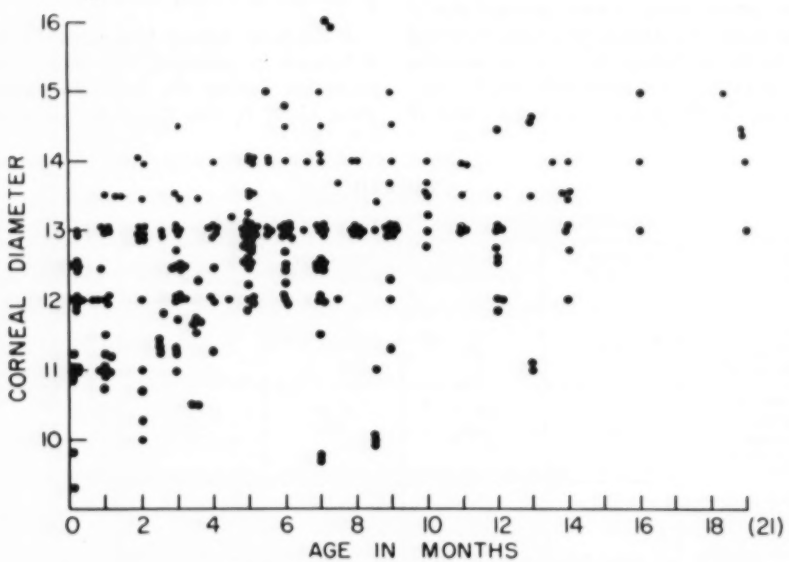

FIG. 2—Relation of age to corneal diameter.

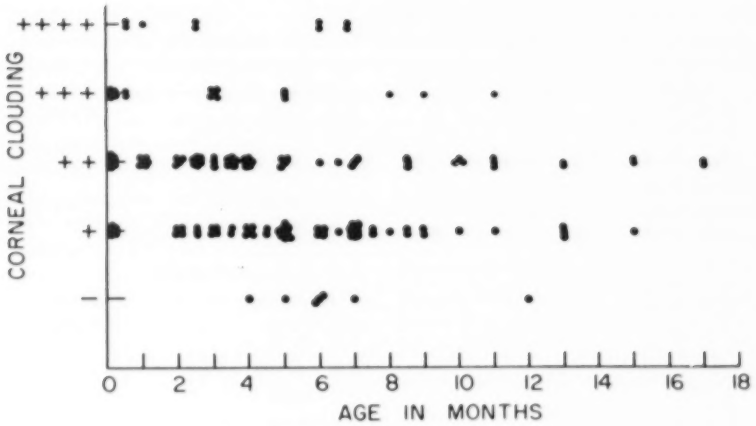

FIG. 3—Relation of age to corneal clouding.

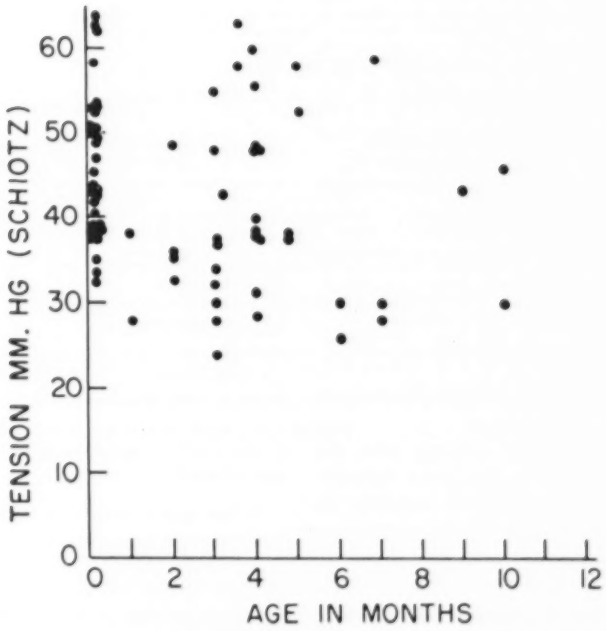

FIG. 4—Relation of age to initial tension.

TABLE IV

VISUAL ACUITY OF FORTY EYES WITH TENSIONS NORMALIZED BY GONIOTOMY

VISION	FOLLOW-UP	VISION	FOLLOW-UP
R.E. 20/60 L.E. 20/100	19 years	R.E. 20/20 L.E. 20/20	8 years
R.E. 20/100	19 years	R.E. H.M. L.E. 20/20	7 years
R.E. 20/50	19 years	R.E. 20/20 L.E. 20/20	7 years
R.E. 20/50 L.E. 20/50	14 years	R.E. 20/20 L.E. H.M. (amblyopia)	7 years
L.E. 20/200	13 years	L.E. 20/30	7 years
R.E. 20/50 L.E. 20/200	12 years	R.E. 20/60 L.E. 20/20	7 years
R.E. 20/70 L.E. 20/40	12 years	R.E. 20/200	7 years
L.E. 20/80	12 years	L.E. 20/30	6 years
R.E. 20/100 L.E. 20/100	11 years	R.E. 20/20 L.E. H.M.	5 years
R.E. 20/30 L.E. 20/30	11 years	R.E. 20/50 L.E. H.M.	4½ years
R.E. 20/200	9 years	L.E. 20/100	4 years
R.E. 20/30 L.E. 20/60	8 years	R.E. 20/20 L.E. 20/20	3 years
R.E. 2/100	8 years		

TABLE V

RESULTS OF THE GONIOPUNCTURE AND THE COMBINED OPERATION

OPERATION	NO. OF EYES	SUCCESSFUL	PER CENT SUCCESS
Goniopuncture	17	10	64
Combined goniotomy and Goniopuncture	22	15	68

TABLE VI

MISCELLANEOUS OPERATIONS FOR CONGENITAL GLAUCOMA

OPERATION	NUMBER	SUCCESSFUL	SUCCESSFUL WITH MIOTICS
Paracentesis	4	0	0
Trepanation	6	0	0
Iridencleisis	11	4	1
Cyclodialysis	5	0	0
Cyclodiathermy	2	0	0

It is suggested by analogy with the goniotomy operation that these successful goniopunctures should continue to function indefinitely. This inference may be premature, however, for it is generally believed that the goniopuncture operation functions as a filtering procedure, and as such, our knowledge of the final outcome must await the passage of time.

When goniotomy was combined with goniopuncture as a primary procedure in 22 cases, the tension in 15 was normalized by one operation. When it was repeated in two eyes, it failed to succeed (table V).

Miscellaneous Operations

Thirty eyes in this series received other forms of antiglaucomatous surgery as the primary procedure. These operations included paracentesis, trephining, cyclodialysis, cyclodiathermy, and iridencleisis. Iridencleisis succeeded in four instances, but the other operations were all failures. The four successful iridencleises resulted in a corrected visual acuity of 20/100 after fourteen years in one case, 20/130 after nine years in another, 20/100 after eight years in still another, and 20/200 after five years in the last (table VI).

In nine cases, cyclodialysis was repeated when the first operation failed, and in no instance did the second operation succeed. In those cases in which a filtering operation was performed, the operation was not repeated, presumably because of technical difficulties.

Secondary Operations

One hundred thirty-three multiple mixed operations were performed. A chart has been prepared which reveals the results of the various antiglaucomatous procedures when they follow procedures of a different type (table VII). It is hoped that this chart will prove of help when similar clinical situations arise.

TABLE VII

SECONDARY OPERATIONS FOR INFANTILE GLAUCOMA AND THEIR RESULTS *

PRIMARY OPERATION	SECONDARY OPERATIONS						
	TREPANATION	IRIDENCLEISIS	CYCLODIALYSIS	GONIOTOMY	GONIOPUNCTURE	COMBINED	CYCLODIATHERMY
Trepanation			2 (—)	11 (+) 3 (—)	1 (—)		1 (—)
Iridencleisis			3 (—)	2 (—) 3 (+)			
Cyclodialysis	2 (—)	2 (+) 1 (—)	5 (—)	6 (+) 3 (—)			2 (—)
Goniotomy	2 (+)	4 (+) 9 (—)	2 (—)		1 (+) 11 (—)	3 (+) 4 (—)	3 (+) 14 (—)
Goniopuncture		2 (—)	1 (—)		1 (+) 1 (—)	2 (+) 5 (—)	5 (—)
Combined		1 (—)		1 (+) 1 (—)	1 (+) 6 (—)	2 (—)	2 (—)
Cyclodiathermy				2 (+)			

* (+) Tension controlled below 28 mm. Hg Schiøtz
(—) Tension uncontrolled

DISCUSSION

This study reveals that the increased tension of infantile glaucoma is normal-

ized by angle surgery in 77 per cent of the cases. This figure is markedly higher than that reported by Anderson,[1] who stated that filtering operations normalized the tension in from 40 to 50 per cent of the cases. Our limited data on iridencleisis are in close agreement with Anderson's figures (fig. 5).

Of interest is the poor prognosis associated with infantile glaucoma present at birth or developed during the first two months after birth. Whether the high tensions, the greater corneal clouding, and the poorer prognosis at this period indicate that this form of glaucoma is a more severe form of the disease seen in the older age groups, or whether these factors are due to a different mechanism, can only be contemplated.

An attempt to analyze the outcome of this disease on the basis of central visual acuity was inconclusive because the initial visual acuity cannot be determined, and because a large number of children with normalized tension are still too young for an adequate postoperative evaluation to be made. When the final outcome is available, it is the panel's opinion that the potential visual acuity will depend on the severity of the disease and the length of time before tension normalization, as well as upon the character of the operative technique. Preliminary results have made possible a markedly better prognosis. In unilateral cases the possibility of amblyopia ex anopsia must not be overlooked.

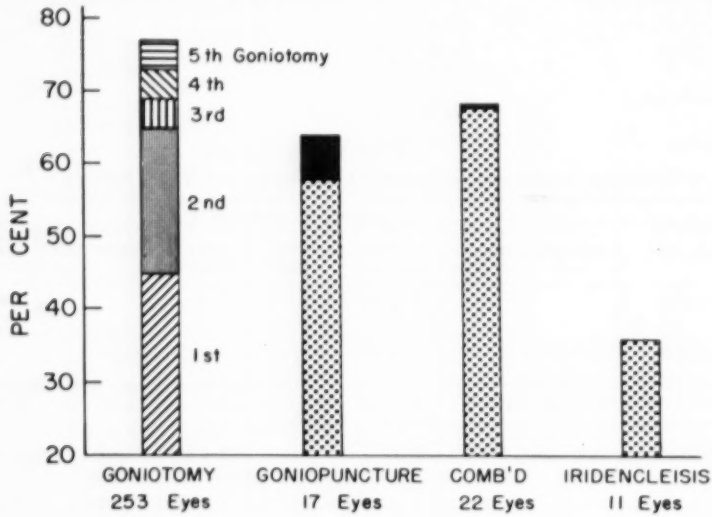

FIG. 5—Percentage of success of various operations for infantile glaucoma.

Since the ophthalmologist cannot control the severity of this disease, it is evident that he must concentrate upon early diagnosis and immediate tension normalization.

Only one death has occurred as a re-

sult of anesthetics administered to these infants, which attests to the safety of modern intratracheal techniques.

SUMMARY

1. The prognosis for infantile glaucoma present at birth or occurring during the first two months after birth is poorer than when the disease occurs later.

2. The medical treatment of infantile glaucoma is condemned.

3. Surgery of the angle in infantile glaucoma results in a higher percentage of tension normalization than occurs when classical filtering procedures are used.

4. It is predicted that early diagnosis with early surgery of the angle will make possible a greatly improved visual prognosis.

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Symposium: Congenital Glaucoma

REVIEW, SUMMARY, AND CONCLUSIONS

SAMUEL J. MEYER, M.D.
CHICAGO, ILLINOIS

THE purpose of this panel has been to organize and present those facts about congenital glaucoma which seem established and clinically important. We have made no attempt to discuss contemporary theory or controversial issues. Fortunately, the disease is very rare and is encountered infrequently by the average practicing ophthalmologist. The members of this panel have probably treated a larger number of cases than any other group, and they have attempted to provide a summary of fundamental knowledge that might serve as a foundation for future study.

The panel has met on several occasions, necessitating travel from coast to coast, and we are greatly indebted to Drs. Otto Barkan and Robert Shaffer for the privilege of studying a large series of their cases, both preoperative and operative, and for a collection of interesting slides showing the pathology of this type of glaucoma.

Congenital glaucoma (buphthalmos) (hydrophthalmia) is a condition wherein structural abnormalities in the region of the anterior chamber result in a decreased facility of outflow of the intraocular fluids and an increased intraocular pressure. In infancy, the ocular coats are elastic enough to stretch with the increased intraocular pressure, so that the entire eye enlarges. In the adult, a similar increased intraocular pressure produces a marked stretching only in the region of the lamina cribrosa.

The condition has been recognized from very early times (Saint-Yves, 1722) (Mackenzie, 1830). Its glaucomatous aspect was probably first noted by Beger in 1744.

The panel is not in favor of using the terms buphthalmos and hydrophthalmia, but recommends the term congenital glaucoma.

Congenital glaucoma is caused by an anomalous development of the anterior chamber angle. About 70 per cent of the known cases are bilateral. While adult glaucoma affects females and males in the ratio of 3:2, about 70 per cent of infantile glaucoma cases occur in males.

While the defect that produces infantile glaucoma is usually congenital, the disease may not become evident for some time after birth.

PATHOLOGY

In specimens of early congenital glaucoma, the *iris* usually is inserted directly into the trabecula, well anterior to the spur, and may cover one half, or more, of the trabecula. Such an anterior insertion could result in a definite block to aqueous outflow. Successful goniotomy may result in a definite recession of the anteriorly placed iris.

Doctor Shaffer believes that an anterior attachment of the iris is usually

From the Department of Ophthalmology, Chicago Medical School, and Michael Reese Hospital, Chicago, Ill.

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present. Gonioscopically, the anterior chamber appears to be deeper. This discrepancy may be caused by gonioscopic prismatic optical changes, which amount to about five diopters of deviation. On the contrary, Doctor Scheie has observed that the angle wall is wide open and that Schlemm's canal is anterior to the spur. He has not seen any anterior insertions of the iris.

The *scleral spur* is less prominent than in the normal eye and is usually situated behind the angle recess. Seefelder believes that the inner lamellas of the trabecula are more compressed in eyes with congenital glaucoma than in normal eyes, and that there may also be an abnormal insertion of the trabecula into the ciliary body rather than into the scleral spur. It is also probable that there may be some defect in the permeability of this tissue. However, the members of this panel have examined the normal eyes of many infants, which have revealed similar findings.

Schlemm's canal is usually found to be poorly developed, or entirely absent, upon microscopic examination. Surprisingly, it is easy to fill Schlemm's canal with blood after pressure normalization, as is frequently observed gonioscopically. The canal may be posteriorly placed (fetal). Some investigators believe that new tissue, either fibrous or endothelial, may be found in the angle. The members of the panel disagree with this belief. Since we can see a clear blood-filled Schlemm's canal gonioscopically, the obstruction to outflow must lie in tissue situated in front of Schlemm's canal.

Doctor Barkan believes that the tissue between Schlemm's canal and the anterior chamber is gonioscopically less transparent. This finding combined with the results of goniotomy has led him to infer that the tissue is less permeable.

DIAGNOSIS

In a large series of cases, Anderson found that the incidence of congenital glaucoma varied from 0.03 per cent to 0.079 per cent of all clinic patients. In institutes for blind children, congenital glaucoma was reported to be the cause of blindness in from 2.4 per cent to 23 per cent. The exact time of onset may be difficult to ascertain, as congestive symptoms may first occur some time after birth. It is usually apparent, however, by the time the child is one year old. The disease is probably present at birth in 30 to 60 per cent of cases, and it may be well advanced. Photophobia, blepharospasm, and epiphora constitute the earliest symptoms of the disease. Photophobia is usually the first symptom, and its presence at birth or shortly thereafter usually indicates the presence of an elevated tension.

The late objective signs consist of enlargement of the globe with an increase in the corneal diameter, usually 12 mm. or more, ruptures of Descemet's membrane, deepening of the anterior chamber, and corneal haze of varying degree. The corneal haze usually fluctuates with the degree of elevated tension present. Ruptures of Descemet's membrane are probably due to the corneal enlargement and stretching. Glaucomatous excavation may occur later, but its presence is difficult to ascertain due to the corneal haze, vitreous opacification, and lens changes.

The intraocular tension must be carefully measured under deep intratracheal anesthesia to obviate an abnormal reading in a normal eye resulting from a struggling child under light anesthesia.

Various investigators believe that gonioscopy is a valuable diagnostic aid and describe persistent mesodermal or anomalously differentiated tissue or abnormal trabecular tissue in the angle as characteristic of the disease. Considerable expert experience is necessary to in-

terpret the pigment layer arrangement at the insertion of the iris into the scleral spur as well as a filled-in apex at the angle of the anterior chamber in infantile glaucoma.

PROGNOSIS

In the past, the prognosis for congenital glaucoma has been very poor. However, as Haas has shown in his statistical report, the outlook for useful vision has improved dramatically. Most of this improvement has occurred since 1939, over a period of 15 years, and is coincident with the popularization of goniotomy by Otto Barkan. It is also possible that with the addition of goniopuncture to our operative armamentarium, especially in cases where goniotomy has failed, the percentage of successful results will increase even more.

If the tension is not successfully controlled, there is a slow progression of the classical signs previously described. The corneal diameter becomes greatly enlarged and the corneal opacification increases. The ocular tissues thin out, and rupture of the globe may occur. Occasionally a spontaneous regression may occur with subsidence of previous objective changes.

TREATMENT

The treatment of congenital glaucoma is primarily surgical. The consensus of the members of this symposium is that deferment of surgery for medical therapy does nothing but deprive the patient of the better result which might be anticipated from early surgery. It is also the opinion of the members of this panel that goniotomy is the operation of choice, an operation which has gained favor as a result of the outstanding work of Barkan, who has reported successful normalization of tension in over 80 per cent of 210 cases operated upon. Goniopuncture, recently described by Scheie, either alone or in combination with goniotomy, also shows some promise in the treatment of congenital glaucoma.

In performing goniotomy, preliminary removal of the clouded epithelium with a keratome makes it possible to operate under gonioscopic control in most cases. If corneal cloudiness is so marked that visualization of the angle is not possible after removal of the clouded epithelium, good results may still be obtained by operating without visualization.

Diamox, which is successful in reducing the intraocular tension and clearing the cloudy cornea, may be tried as a temporary expedient, but this form of therapy should not be allowed to delay the operation.

The sooner pressure is relieved, the greater is the reversibility of the corneal cloudiness. Goniotomy while the disease is in its early stages also enhances the opportunity of activating the potentially functioning Schlemm's canal mechanism, which is usually present but which atrophies if aqueous is not given access to it at an early date. A small angle area, if cleanly exposed, is often sufficient to normalize the intraocular tension permanently. It is possible that only a small area of exposure suffices for physiologic demands, provided that the exposed area is normally permeable and has not become scarred as a result of surgical trauma. The action of goniotomy probably consists of re-establishing outflow to the trabecula-Schlemm's canal mechanism and its emissaries.

Goniotomy is contraindicated in the late distended stage of infantile glaucoma in which Schlemm's canal has become more or less obliterated. During that stage, the operation may cause a sudden loss of intraocular fluid with a resultant retinal detachment or dislocation of the lens. In late cases, cyclodia-

thermy punctures and oral use of diamox may result in some degree of success.

Recently, some European ophthalmologists have reported a series of successful results in controlling the tension by using cyclodiathermy. Such favorable results have not been corroborated by the members of this symposium.

Results from other procedures, such as corneoscleral trepanation, iridencleisis, and cyclodialysis, in the hands of this committee have been very discouraging. Use of these operations also accounts for the poor results reported by all investigators previous to 1939, when goniotomy was first brought to our notice. Corneoscleral trepanation rarely controls the tension adequately, and all too frequently it may be accompanied by loss of vitreous, intraocular hemorrhage, subluxation of the lens, corneal edema, and permanent corneal opacity.

It should be noted that the use of miotics, diamox, or both, if successful in controlling the tension, should not result in a postponement of surgery. These medicaments are merely an aid to operative measures and to controlling the intraocular tension between operations, when further operation is indicated. It seems that the better the condition of the eyeball at the time surgical measures are instituted, the better chance there is for controlling the tension. It is difficult to ascertain the amount of vision present following operative interference, due to the young age of the individual affected, and to the short period of time in which the postoperative results of goniotomy and goniopuncture have been under observation. It is probably best to rate the percentage of successful results according to the number of cases with normalized intraocular pressure. When the tension is normalized, the corneal haze clears, and probably no further deterioration of the eyeball occurs.

No doubt the percentage of eyes with normalized tension following goniotomy or goniopuncture will improve materially with the increase in experience gained by the occasional operator. This is adequately demonstrated by the contrast between the indifferent results reported on a series of cases done by ophthalmologists not very experienced in angle surgery and the results in a series of cases performed by more experienced and capable surgeons. The statistics reveal emphatically that the inexperience of the operator will influence adversely the over-all percentage of cures. The more angle surgery performed, the greater improvement in the ultimate results.

Early surgical normalization of intraocular pressure is needed to prevent marked corneal enlargement and stretching of the other ocular tissues, with the secondary degenerative and irreversible changes that may follow, in order to allow development of the central visual acuity.

In conclusion, the members of the symposium believe that early diagnosis and surgical intervention are indicated. Goniotomy is the operation of choice. Goniopuncture in combination with goniotomy is indicated if goniotomy is unsuccessful. Diamox may be used temporarily to lower the tension preoperatively.

SUBACUTE CIRCUMSCRIBED MACULAR RETINOCHOROIDITIS SIMULATING INTRAOCULAR TUMOR

W. E. Fry, M.D.
Edmund B. Spaeth, M.D.
PHILADELPHIA, PENNSYLVANIA

This paper concerns the ophthalmoscopic and microscopic appearance of subacute circumscribed retinochoroiditis of the macular area. The clinical appearance of this type of retinochoroiditis will be compared to the clinical appearance of an early case of macular malignant melanoma, with reference to the similarities and the differences in appearance.

The pathologic appearance will be described and compared to the pathologic changes seen in a type of nonspecific inflammatory change involving the retina and the choroid. The report is based on five cases, three of which have pathologic reports associated with them.

CASE REPORTS

Case 1—E. G. D., age 27, a white male, was admitted to the service of E. B. S. at the Wills Eye Hospital on June 6, 1949.

The vision on admission was 6/6 in the right eye and 2/60 in the left eye. The Schiøtz tension was 17 mm. Hg in each eye.

The chief complaint on admission was diplopia followed by loss of vision in the left eye. The patient first noticed diplopia about January 15, 1949. It was present in all fields of vision and increased over a short period of time. Following this there was a gradual loss of vision. There was no previous history of ocular trauma or disease. The patient had worn glasses at no time. The previous medical history was unimportant except for malaria in 1943, with several recurrences, and an attack of pyelitis in 1942.

Previous to the time of admission to the Wills Eye Hospital the patient had had a series of penicillin injections, and later on a series of penicillin injections combined with streptomycin. There had been no change in the fundus condition following this treatment, which extended over a period of eight weeks.

All reports on the general physical examination were normal. The patient was a healthy adult male. Blood pressure, 120/75 with normal physical findings.

The external condition of the eyes was normal. There was no injection of the conjunctiva, the corneas were clear and the pupil reaction was perfectly normal. The rotations were full. In the fundus of the right eye, media were clear and the disc was normal in size, shape, color and contour. The vessels were normal; macula and periphery were normal.

In the left eye the media were clear. The disc was normal in size, shape and color. The vessels appeared to be of average size and appearance. In the macular area there was a grayish elevated mass, the top of which measured 2 diopters above the general level of the retina at the posterior pole (fig. 1). There were several small hemorrhages about this

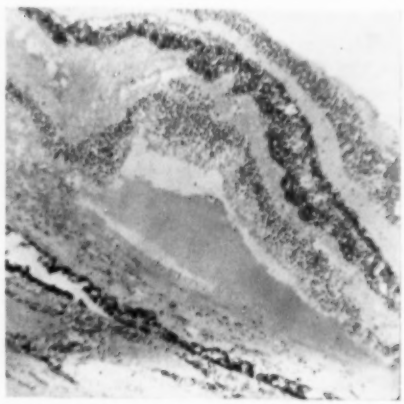

FIG. 1—Case 1. Macular area.

From the Wills Eye Hospital.
Presented at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

area. There was no change in color of the surface as far as irregular pigmentation was concerned.

At a later date the fundus in the left eye was described as showing a few fine vitreous opacities. The disc margin was slightly blurred but with no elevation. There was an area of increased pigmentation between the macula and the disc which extended up and around this like a collar. The lesion did not extend beyond the temporal margin of the macula.

There was one small fresh hemorrhage above the lesion. The retina overlying the lesion appeared edematous and without pigmentation.

The laboratory reports were not illuminating with regard to the ocular situation. Roentgenograms of the skull and sinuses indicated polyps in the floor of the left antrum; the skull, sella turcica, orbits and fissures showed no evidence of disease.

The blood sugar was 78. The spinal fluid examination showed the pressure to be normal. Wassermann and Kahn tests were negative, and the blood count was within normal limits.

The patient was seen by several ophthalmologists in consultation. The eye was enucleated on June 11.

Pathologic report

The gross specimen was a globe of normal size with no abnormality as to contour.

Microscopic examination

Cornea showed slight edema in the epithelial layer but otherwise was not noteworthy.

Anterior chamber was of average depth. The filtration angles were open. The spaces of Fontana were distinct. The iris was normal throughout. There was a slight ectropion of the uveal pigment, the pigment layers showing loss of pigment to some extent.

Ciliary body: There was a slight pigmentary loss but no other pathologic change. The zonular fibers were intact. The lens appeared normal. Vitreous chamber was free of exudate.

Retina and choroid: The retina in the macular region was edematous and thrown into folds. The folds were deep and involved all of the layers of the retina (fig. 2). The retina was separated from the underlying choroid, and a granular exudate type of material was present in the deeper layers of the retina. The layer of cones at the macula had disappeared and this area was replaced by an exudate containing round cells as well as granular material, with some red cells. Slightly

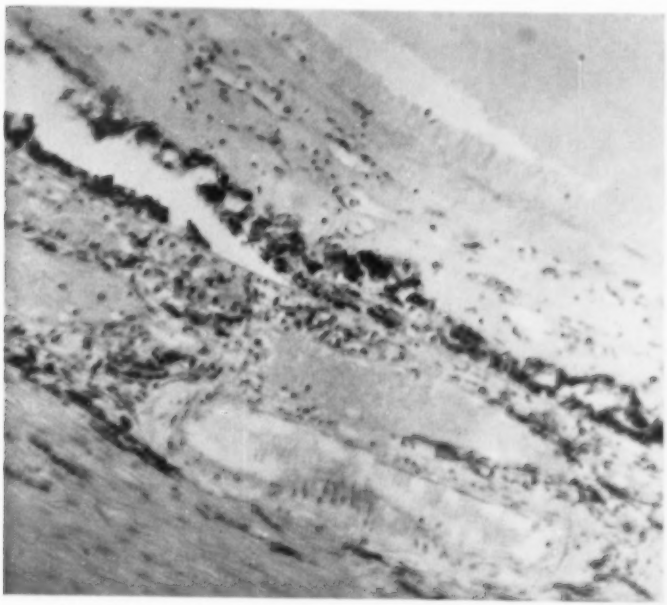

FIG. 2—Case 1. Macular area showing retinal pigment epithelial change.

to the disc side of the macular region there was some proliferation of cells resembling the elongated type of cell that is derived from the pigment epithelium. The pigment epithelium could be traced beneath the whole lesion; it was quite irregular but it was present in most areas. Bruchs' membrane could be made out in most places. The choroid beneath this area was approximately twice the normal thickness and contained dilated blood vessels as well as a moderate amount of round cell infiltration. Some of this round cell infiltration could be traced to the edge of the optic nerve. The retina and choroid away from the macular region appeared normal except for a localized proliferation of pigment epithelium near the periphery at one side. This was unassociated with a round cell infiltration.

Impression

A moderately early stage of subacute retinochoroiditis.

Case 2—These findings illustrate a slightly different phase of the condition under consideration. This case has been reported previously but will be reviewed briefly at this point because the pathologic findings indicate a more advanced stage of the disease than those of case 1.

History

D. M., age 30, male, white, admitted to the Wills Eye Hospital on June 18, 1949, under the service of W. E. F.

The chief complaint was poor vision of the right eye for a period of several months. The patient stated that his vision in the right eye was known to be normal by examination in 1945. He had never worn glasses. His vision on admission was 1/60 in the right eye and 6/6 in the left eye.

The previous medical history contributed nothing of value in diagnosis of the present condition.

General physical examination revealed a well nourished, well developed white male without abnormal physical findings. The blood pressure was 130/80.

External ophthalmoscopic findings were normal in both eyes. The intraocular tension was 19 mm. Hg Schiøtz in each eye.

Fundus examination: O. D., media clear, disc normal in size, shape, color and contour with a normal physiological cupping. The vessels were normal in caliber. The macula showed an elevated area, approximately 2 diopters in height (fig. 3). It was densely pigmented, and lateral illumination showed a moth-eaten type of central spot. There were a few hemorrhages on the periphery of the elevation, and a zone of serous exudation surrounded it. A small amount of yellowish exu-

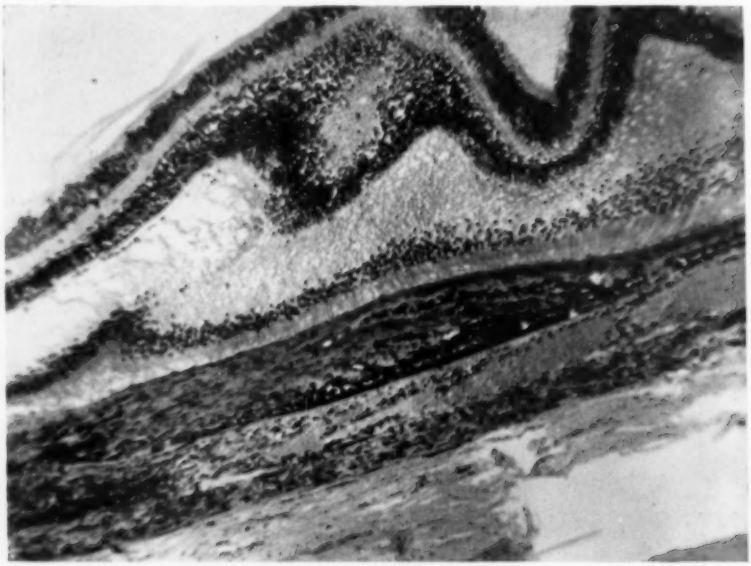

FIG. 3—Case 2. Macular area.

date appeared approximately 1.5 disc diameters temperoinferiorly from the macula. The periphery of the retina was otherwise normal.

O. S., media clear, disc normal, vessels normal and macula normal. Retinal periphery normal.

The slit lamp findings were normal in each eye.

The laboratory reports, including blood count, tests for blood sugar and blood urea, Wassermann and Kahn tests, and Bence Jones protein test, were entirely normal. Roentgenograms, including those for chest, bones and skull, gave normal findings. The patient was discharged following the first admission but was readmitted again August 8, 1949. At that time there had been an increase in the size of the macular lesion in the right eye; the elevation remained the same, but the size of the scotoma in this eye had increased. There were consultations with several ophthalmologists. Enucleation was carried out on August 13, 1949.

Pathologic report

Corneal structure was normal. The anterior chamber was of normal depth. Iris showed edema of the pigment layer with some loss of pigment. Posterior synechias were present on both sides of the pupillary opening. The lens was normal. The ciliary body showed pigment migration.

The choroid and retina: The retina was thrown into folds to a moderate extent; it was not separated from the underlying tissue. There was a moderate amount of edema, particularly noted in the inner nuclear layer of cells. Just beneath the retina was an elongated area, about half of the retinal thickness, that was made up of a fibroblastic type of cells similar to those seen as the result of proliferation of pigmented epithelial cells (fig. 4). There was no inflammatory exudate in this area, but at one side there was a small collection of red blood cells. Beneath this area was some irregular pigmentation from the pigment epithelial cells, and still further beneath this area of pigment epithelial cells was an area of inflammation of the choroid. In this portion the choroid was about twice the normal thickness and contained a moderate number of small round cells. For the most part these were widely separated and in only one area did they form a definite collection. The area between the macular lesion and the optic nerve appeared uninvolved except for one point just adjacent to the optic nerve, where there were some adhesions between the deeper layers of

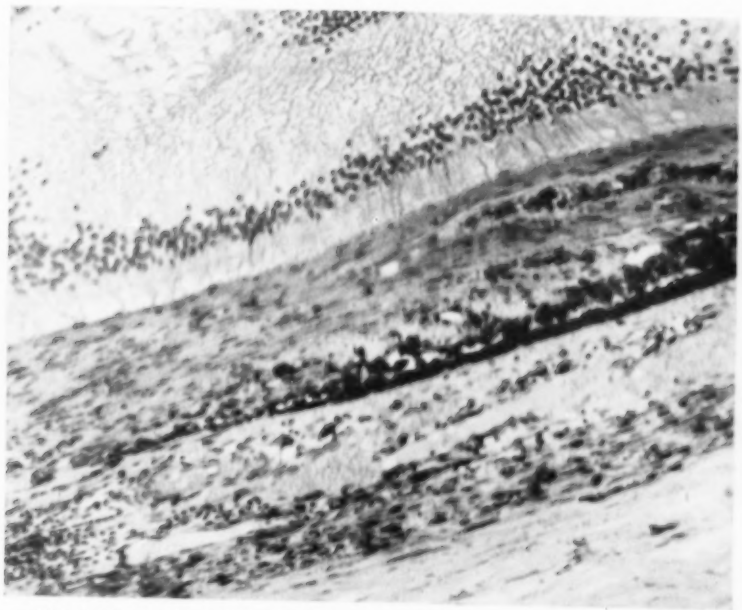

FIG. 4—Case 2. Macular area showing proliferation of retinal pigment epithelial layer.

the retina and the pigment epithelium. The retina and the choroid away from the posterior pole appeared to be normal.

Impression

Sub-acute localized retinochoroiditis. This represents a later stage than that in case 1.

Case 3—C. N., age 45, white, male, seen in the office of W. E. F. on February 17, 1954.

Approximately two years ago the patient was aware of a loss of central vision in the left eye. Objects became blurred and were wavy and distorted. The condition became slowly worse over a period of a number of months. During the past year the situation and vision in the left eye has remained stable, although the patient believes that his vision at the present time is slightly better than it was six months ago.

At the time of the beginning of his ocular difficulty, general physical survey and laboratory studies indicated that the patient's health was normal.

Visual acuity in the right eye was 6/6+, and in the left eye, 1/60. The central fields of the right eye were normal. The central field in the left eye showed a central scotoma for 2/1000 white, approximately 8 degrees in diameter.

The external ocular condition was entirely normal. The fundus of the right eye was noteworthy. The disc was healthy, the macular region was normal. In the left eye, the lens and vitreous were clear, the disc was healthy and the vessels showed no change. In the macular region was a central white area approximately one disc diameter in size with a collar of pigmentation (fig. 5). There were no hemorrhages. Directly in the central area there was a lesion with yellowish white exudate; the top of the lesion measured 2 diopters.

FIG. 5—Case 3.

Impression: Stabilized circumscribed subacute retinochoroiditis.

Case 4—W. N., age 86, white male, admitted to the service of W. E. F. at the Wills Eye Hospital, June 12, 1953.

There was a history of gradual failing of vision in the left eye over a period of seven years. The patient had had no treatment. There had been no light perception with this eye for at least six months prior to the time of admission.

Findings from the physical examination were normal. The ocular examination of the right eye showed no light perception; tension as tested by the fingers was normal. Some corneal stain appeared in the lower one third; the anterior chamber was poorly visualized because of the hazy cornea. The iris showed no uveal vascularization; posterior synechias could not be discerned.

The lens showed a central opacity; the fundus could not be seen.

Pathology

Cornea: The epithelium was intact, as in Bowman's membrane. The stroma appeared to be somewhat more cellular than normal with irregular distribution of the cells. Descemet's membrane was intact. The anterior chamber was of moderate depth.

The angles were occluded. The iris appeared to be somewhat more cellular than normal. There were posterior synechias. The ciliary body showed no inflammatory change. The crystalline lens was intact.

Both the retina and the choroid appeared somewhat thinner than normal in most areas. At the posterior pole, however, on both sides of the optic nerve the retina shows marked degenerative changes. The unusual feature, however, was a considerable proliferation of cells, similar to the pigment epithelial proliferative cells, between the retina and the choroid. This occurred on both sides of the optic nerve and was unassociated with an inflammatory exudate or with hemorrhage (fig. 6). The optic nerve was notably affected.

Impression

This represents a nonspecific, moderately diffuse retinochoroiditis limited to the posterior pole but not limited to the macular region. The changes in this case seem to be, in our opinion, much more advanced than those in the first two cases, but the process appeared to be a similar one.

Case 5—O. L., age 44, white female, seen

FIG. 6—Case 4. Retinal and choroidal change adjacent to the optic nerve.

in the office of W. E. F. on November 13, 1953.

The visual acuity in the right eye was 3/60 and in the left eye 6/6. The patient stated that she first noticed two days before that the vision in her right eye was blurred. There was a history of a fall down steps eight weeks previously. At that time her wrists were badly bruised and she had a nosebleed, but at the time no damage to her eyes was noted subjectively.

The eyes were entirely normal externally. The pupils were equal and reacted normally. The intraocular tension was normal.

In the fundus of the right eye a grayish mass began in the macular region and extended temporally for a distance of 2 to 2.5 disc diameters. The surface of this mass was elevated 6 diopters. It was grayish brown in color with some pigment mottling in and beyond the macular region. There were no hemorrhages. There were signs of pigment mottling just beneath the surface of the mass, and some areas had a yellowish appearance.

The slit lamp examination in both eyes was entirely normal. The right eye was enucleated on November 16, 1953.

Pathologic examination

The anterior segment was entirely normal. The angles were open, the iris appeared normal and the crystalline lens was normal. Beginning in the macular region and extending

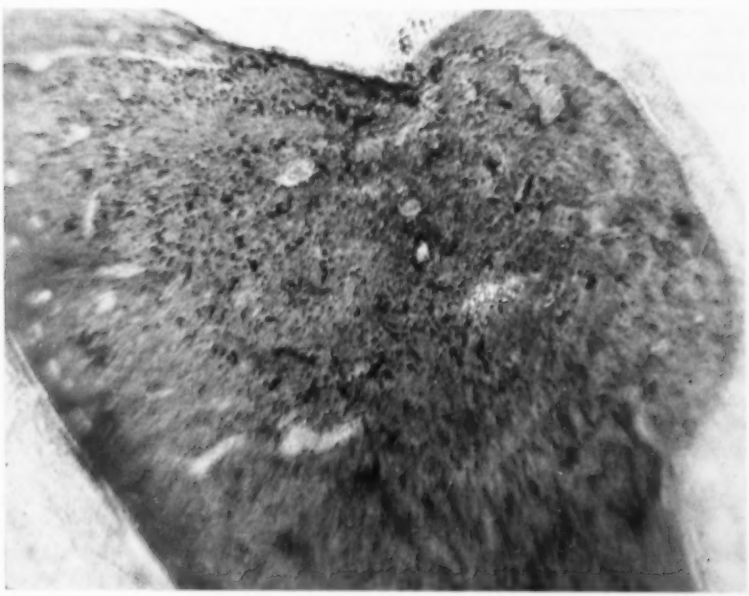

FIG. 7—Case 5. Malignant melanoma in macular area.

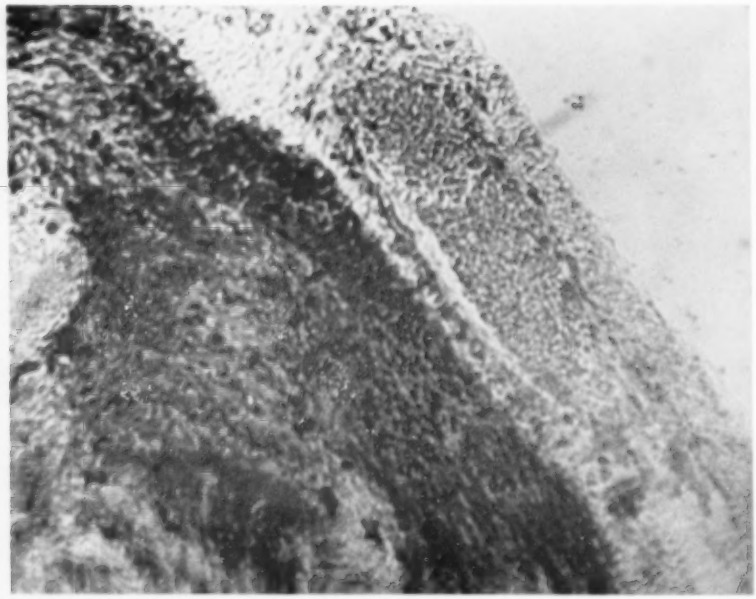

FIG. 8—Case 5. Apex of tumor.

out approximately 4 millimeters was an elevated pigmented tumor-like mass made up of spindle cells (fig. 7 and 8). The diagnosis was spindle-cell malignant melanoma.

There are certain clinical features of these cases of localized retinochoroiditis and tumor formation at the macula that may be compared.

The vitreous was clear in both conditions. The anterior chamber and cornea showed no unusual changes on slit lamp examination. There was no iris vascularization in the cases of retinochoroiditis. We have noted no iris freckles in the case wherein the malignant lesion was located at the posterior pole. The disc was normal in both instances and the retinal vessels were not contributory. Many of these cases occur in young individuals and the situation is not necessarily associated with hypertension. In both instances the visual acuity may be 6/60 or less. In the cases of localized retinochoroiditis the symptoms were loss of vision, some distortion, and frequently a history of aching. One patient described diplopia. In the case of the tumor there was distortion of the image and visual loss. The peripheral fields in all the cases were normal. In the cases of retinochoroiditis, there was an irregularly rounded scotoma in the central field. This varied in size depending upon the activity of the lesion. In the case of the macular tumor, the scotoma varied in extent and showed a tendency to increase in size as the condition advanced. The ophthalmoscopic appearance of the lesion in the case of macular choroiditis was somewhat hazy in outline, and the area of involvement was usually not greater than 1 and 1.5 disc diameters in extent. In the case of the macular tumor, the outline was also hazy. However, the size of the tumor may vary considerably depending upon the extent and duration of the lesion. In the cases of retinochoroiditis, the eleva-

tion was less than 3 diopters above the level of the surrounding retina. With a possible macular tumor, the elevation may vary, but even when the tumor is first under observation the size of the elevation may be from 6 to 8 diopters. In case of macular choroiditis, hemorrhages frequently form a partial collar during the early stages, but later on they clear up; while with a tumor, the hemorrhage is absent. Macular choroiditis frequently gives a grayish appearance to the retina, and at times there may be a central yellowish spot. In the tumor case the color of the retina was gray or greenish gray. The pigmentation varied; with the macular choroiditis in the early stages it was minimal in extent. Frequently the more advanced the lesion, the greater the amount of pigmentation. In the case of tumor the pigment disturbance was minimal. The active period of macular choroiditis may vary from three to nine months in most instances, with some extension of the time in unusual cases; with the tumor the elevation was definitely established when it first came under observation and from the history the progress appeared to be rapid. In the cases of retinochoroiditis the lesion tended gradually to flatten out, whereas the tumor increased in size.

There are certain pathologic changes that can be compared with advantage. The anterior segment was normal in both the cases of retinochoroiditis and the tumor. The vitreous was clear in both instances. In the cases of retinochoroiditis, there were a few round cells at the margin of the disc at the level of the choroid; in the case of tumor the disc was normal. In the cases of retinochoroiditis, the retina was elevated and thrown into folds. The degree of edema varied, with separation of the layers and with some collections of round cells. There was a collection of red cells at the edge of the lesion. With the tumor the retina was elevated; there was no unusual change in the architecture of the various layers and hemorrhage was absent, but dilated vessels were at the margin and on the surface of the growth. In central retinochoroiditis there was proliferation of the pigment cells with scattering of the pigment granules, and a varying amount of fibroblastic proliferation accounted for much of the tumor-like appearance. This fibroblastic proliferation was sharply limited to the macular region. The pigment epithelium over the mass of the tumor showed no unusual change. Bruch's membrane was unbroken in the cases of retinochoroiditis. It stained quite well, indicating some thickening. In the tumor case, Bruch's membrane had ruptured so that the tumor bulged through into the subretinal space. The choroid in the cases of retinochoroiditis was thickened, the vessels were dilated and there were scattered collections of round cells. These changes were limited to the macula and the immediate surrounding areas. In the case of tumor, the mass could be seen to expand the choroid with proliferation of the tumor cells in this area. The vessels at the edge of the tumor were dilated.

Certain comparisons can be made between the changes that occur in the localized macular retinochoroiditis and in the diffuse type. In both instances the pigment epithelial cells showed a fibroblastic proliferation that cannot be distinguished from the fibroblastic proliferation that was present in the case of diffuse retinochoroiditis of unestablished etiology.

A practical observation can be made from the pathologic findings in these cases of macular disease: the process appears to be an inflammatory one and nonspecific therapy should be used vigorously, especially in the early stages. Nonspecific therapy is usually all that

can be given because careful investigation for a specific etiology is disappointing in an overwhelming percentage of the cases.

SUMMARY

1. Two cases have been described, illustrating the subacute inflammatory nature of cases of localized retinochoroiditis that simulate an intraocular malignant growth.

2. The clinical and pathologic findings in a case of malignant melanoma of the macula have been described.

3. The differences in these two conditions have been pointed out.

4. The end result of a case of localized retinochoroiditis has been presented.

5. The pathologic changes in localized subacute macular choroiditis appear to be similar to those in a case of diffuse retinochoroiditis of undetermined or varying etiology.

6. The importance of active therapy in the early stages of macular disease has been indicated.

DISCUSSION

BERTHA A. KLIEN, M.D., Chicago, Ill.: The differential diagnosis of elevated, pigmented macular lesions is one of the most difficult in ophthalmology, one of the most critical for the patient, and one which, sooner or later, confronts every ophthalmologist. An analysis of a group of cases of this kind, with the correlation of clinical and histopathologic findings whenever it was posible, such as Doctor Fry and Doctor Spaeth have presented today, is a real contribution to the solution of this difficult problem.

I would like to re-emphasize some of the points they made and add a few observations.

This problem is made more complicated by the fact that several conditions other than inflammatory lesions have to be differentiated from early choroidal tumors of the posterior polar region, viz., those of degenerative and functional origin, all of which may produce elevated and pigmented lesions.

The significant findings in this differential diagnosis may be divided into negative, those which speak against a tumor, and positive, those which speak for a tumor. The negative findings outnumber the positive.

Among the negative findings are the following:

1. The presence of intraretinal or subretinal hemorrhages. Such hemorrhages are common in the inflammatory, degenerative and functional lesions, but they are absent in early choroidal tumor. There may be rare exceptions when they do occur, but I have seen none. In two of the authors' cases of subacute macular chorioretinitis, hemorrhages were noted.

2. The presence of retinal traction folds, which may be radial or concentric to the lesion. The radial folds are freqent in inflammatory conditions, when the inflammation is present as well as in the healed stage. In the active stage they arise because of cellular infiltration of the retina, which tenses the surrounding normal tissue and draws it into folds. Traction folds in the healed stage are due to shrinkage of fibrous scar tissue. The concentric folds are found in edematous imbibition of the macular retina of inflammatory or angiospastic origin, and are always an indication of an intraretinal disease process, such as is not found in early choroidal tumor (demonstration of kodachrome slides).

3. A third negative, but very useful, finding is the increasing distinctness of the outline of a lesion in spite of an increase in size objectively, as well as an increase in the size of the central scotoma subjectively. This occurs in the healing stage of inflammatory processes, also in degenerative macular lesions and in residual findings of functional disturbances.

4. A fourth group of negative findings might be called incidental or accessory findings, because they are less common than those mentioned above but, if present, speak against a tumor, viz., lipoid infiltration of the retina, which indicates an intraretinal process of some standing, such as is not associated with tumor. The fundus description of Case 2 in the paper just read is suggestive of such a lesion.

Another symptom, equally useful, is diplopia, preceding or simultaneous with the development of the visual disturbance, such as was present in the authors' first case and in the second clinical case I demonstrated. It certainly suggests similar processes in retina and brain and does not indicate a tumor.

Vitreous opacities, such as were described in the authors' first case, especially if they decrease or disappear again, also speak against a tumor.

The positive findings, which speak for a tumor in the macular region, are few.

1. The increasing blurring of the outline of the lesion with increasing size, due to an expanding retinal separation around it, a finding very suggestive of tumor.

2. The direct visualization of the tumor made possible recently by means of the Haag-Streit slit lamp and the Hruby lens.

Unfortunately, in macular tumors the positive accessory findings, which prove useful in diagnosing tumors in other parts of the choroid, are not helpful. Doctor Fry and Doctor Spaeth have pointed out that iris freckles are not usually associated with tumors of the posterior polar region. Similarly, cystoid degeneration of the retina overlying the tumor, which has recently been pointed out by Rones and his associates as a differential diagnostic aid, is not helpful in dealing with posterior polar tumors, since the macular region is a site of predilection for cystoid degeneration in a variety of conditions.

The term "retinochoroiditis," as used by Doctor Fry and Doctor Spaeth, has to be given a rather broad interpretation. While the origin of the lesions in their cases is possibly inflammatory, it need not be, and probably it is not a primary inflammation of the tissues of retina and choroid as such.

It could be a disease of some of the blood vessels supplying this region. In this connection the history of diplopia given by one of the patients is noteworthy again. It reminds one of another vascular disease of young males, viz., Eales' disease, in the course of which diplopia may occur due to transient paralysis of various groups of ocular muscles.

It also could be an inflammatory focus outside of the eyeball which has a toxic effect upon the macular vessels. Transudation, exudation and hemorrhages from the macular vessels thus affected and irritative proliferation of the pigment epithelium would produce the histologic picture shown by the authors. They pointed out the complete lack of specific inflammatory signs. The picture is dominated by the end results of activity of the pigment epithelium (demonstration of kodachrome slides).

It cannot be overemphasized that for practical purposes a low-grade chronic irritation of this most versatile of cellular structures may result in pigmented mounds through simple proliferation, or proliferation with production of large amounts of cuticular substance, or proliferation with metaplasia into fibrous or glial masses, all of which appear clinically as elevated, pigmented lesions.

In closing, I wish to congratulate the authors on the collection of this interesting, though small, group of cases, which should have taught all of us something about the differential diagnosis of macular lesions.

DOCTOR FRY: Doctor Klien has significantly enhanced the presentation of this paper by her illustrations.

One thing might be mentioned. Because of the size of the instrument, P-32 studies were not done on cases of this type. When instruments are devised so that we can obtain P-32 studies on these cases, they will be extremely useful. They will help us a great deal in our clinical evaluation of these situations.

THE EFFECT OF RETROBULBAR INJECTIONS OF PROCAINE ON THE OPTIC NERVE

FRANK D. CARROLL, M.D.
ANDREW DE ROETTH, JR., M.D.
NEW YORK, NEW YORK

DETAILED observations of the effect on the optic nerve of retrobulbar injections of procaine never have been reported so far as we know. One reason is that such injections are usually given preoperatively or for acute glaucoma, and in neither case is it feasible to make accurate studies of visual acuity, visual fields and fundi. A more cogent reason is that it has been generally assumed that the optic nerve is not affected by this procedure. During the past year we have had an unusual opportunity to study this relationship, and our findings have been sufficiently striking to warrant reporting.

At the glaucoma clinic of the Institute of Ophthalmology in New York City an attempt has been under way to measure the effect of the retrobulbar injection of procaine on patients with glaucoma or suspected glaucoma. There is a theory that many of the features of glaucoma not yet understood are the results of neurovascular mechanisms. In the interest of illuminating this area of uncertainty, we thought it might be useful to block the sensory and motor nerves to the eye and then measure the change in intraocular pressure, the rate of formation of the aqueous, and the facility of the aqueous outflow. This is mentioned only to explain why these retrobulbar injections were given. The findings relevant to the initial inquiry have been described in detail in a separate report.[3a] The present paper is concerned solely with the effect of the retrobulbarly injected procaine on the function of the optic nerve.

INVESTIGATIVE PROCEDURES AND RESULTS

In the course of this study a total of 95 retrobulbar injections have been given to patients in the glaucoma clinic. In each case the tonography, visual acuity, visual fields, and appearance of the fundi were recorded prior to injection and at frequent intervals thereafter. The technique of making the injections was as follows: The patient was asked to look up and in; a 35-mm. 26-gauge needle was introduced through the skin of the lower lid temporally and passed over the orbital rim just beneath the globe between the external and inferior rectus muscles toward the optic canal; after an attempt to withdraw the needle, 1 cc. of solution was injected. The patient was then asked to look down and in; another 1 cc. was injected and the needle withdrawn.

A series of 28 eyes in 26 patients has been observed critically for the effect of the retrobulbar injection on the function of the optic nerve, and it is with this group only that we are concerned in the present report. Of the 28 eyes, the condition in 16 had been diagnosed as open-angle glaucoma, and in 4 as shallow-angle glaucoma; 8 were considered normal eyes in which glaucoma had been suspected.

From the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, and the Institute of Ophthalmology, Presbyterian Hospital.

Presented at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

Four of the 26 patients were injected with saline to serve as controls: 2 received 2 cc. saline, and 2 received 2 cc. saline plus hyaluronidase (15 turbidity reducing units per cubic centimeter). There were no changes in the visual acuity or fields of any of these control patients, although one showed transitory ptosis and another developed transitory extraocular muscle paralyses, ptosis and slight pupillary dilation.

The remaining 22 patients were injected with concentrations of procaine varying from 2 to 8 per cent, with and without hyaluronidase, with the following results.

One patient received 2 per cent procaine without hyaluronidase, and 7 patients received 2 per cent procaine with 15 units hyaluronidase per cubic centimeter. The 2 per cent procaine alone caused a temporary decrease in visual acuity from 20/15 to 20/60. The 2 per cent procaine plus hyaluronidase caused a decrease which varied from very slight (20/20 to 20/25) to very marked (20/20 to 20/200) (fig. 1 and table I).

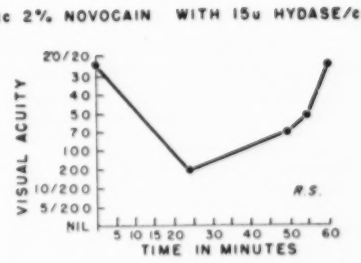

FIGURE 1.

Four patients received 4 per cent procaine with hyaluronidase and sustained a similar reduction in visual acuity, from slight (20/30 + to 20/30 —) to profound (20/30 to 20/200) (table II).

Two patients received 8 per cent procaine without hyaluronidase and both showed rapid and profound loss of vision, i.e., to 3/200 in one, and to no light perception in the other (figs. 2 and 3).

Ten patients received 8 per cent procaine with hyaluronidase. The decrease in vision again varied considerably but

TABLE I

2 PER CENT PROCAINE—EFFECT OF RETROBULBAR INJECTION ON OPTIC NERVE

SOLUTION USED	VISION BEFORE	WORST VISION AFTER	TIME IN MINUTES	DURATION OF EFFECT IN MINUTES	FIELD	% MOTOR BLOCK
3 cc. 2% Procaine	20/15	20/60	12	60	Contraction	50
Procaine & Hyaluronidase 15 u/cc.						
2 cc. 2%	20/30	20/50	6	60	Contraction	70
2 cc. 2%	20/40+	20/200 —	6	49	Contraction	80
2 cc. 2%	20/20	20/30	17	75	Normal	90
2 cc. 2%	20/20 —	20/25	4	19	Normal	0
2 cc. 2%	20/20	20/50	7	60	Normal	80
2 cc. 2%	20/20	20/50	25	60	Normal	90
2 cc. 2%	20/25	20/200	24	60	Unsatisfactory	80

TABLE II

4 PER CENT PROCAINE—EFFECT OF RETROBULBAR INJECTION ON OPTIC NERVE

SOLUTION USED	VISION BEFORE	WORST VISION AFTER	TIME IN MINUTES	DURATION OF EFFECT IN MINUTES	FIELD	% MOTOR BLOCK
Procaine & Hyaluronidase 10 u/cc.						
2 cc. 4%	20/30	20/200	8	74	Contraction	100
2 cc. 4%	20/30	20/60	6	25	Indefinite	30
2 cc. 4%	20/30+	20/30—	10	90	Normal	50
2 cc. 4%	20/30+	20/40—	19	35	Normal	90

was in most cases very marked (figs. 4, 5 and 6). In 8 of the 10 cases the vision became less than 20/200 within a few minutes or in less than 20 minutes, after the injection (table III).

Changes in the visual fields varied widely; there was usually a depression of the entire field. Even when the acuity was reduced only slightly, e.g., from 20/15 to 20/30, the field showed a generalized contraction (fig. 7).

No central scotomas were observed (fig. 8).

Sometimes only a small island of vision remained temporally (figs. 9 and 10) and occasionally, as reported previously, even light perception was lost.

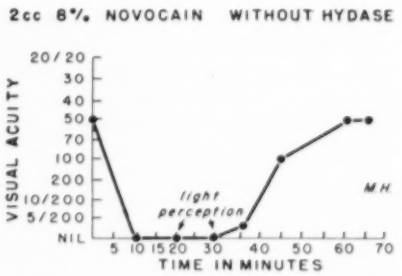

FIGURE 2.

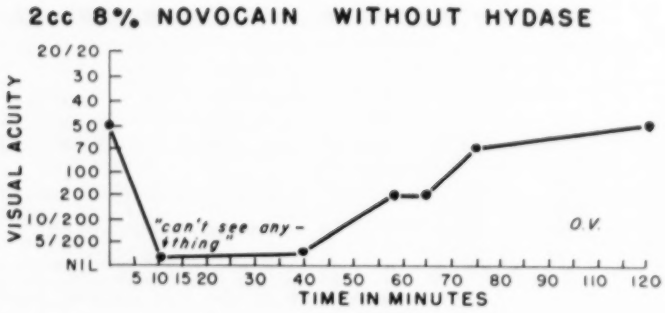

FIGURE 3.

TABLE III

8 PER CENT PROCAINE—EFFECT OF RETROBULBAR INJECTION ON OPTIC NERVE

SOLUTION USED	VISION BEFORE	WORST VISION AFTER	TIME IN MINUTES	DURATION OF EFFECT IN MINUTES	FIELD	% MOTOR BLOCK
2 cc. 8% Procaine	20/50	Fingers at 3 ft.	40	120	Contraction	100
2 cc. 8% Procaine	20/50	Nil	9	120	Nil	95
Procaine & Hyaluronidase 15 u/cc.						
2 cc. 8%	20/25	20/50	20	60	Normal	60
2 cc. 8%	20/40	Fingers at 2 ft.	21	120	Contraction	90
2 cc. 8%	20/70	20/300	13	90	Contraction	90
2 cc. 8%	20/20	Fingers at 1 ft.	10	90	Contraction	90
2 cc. 8%	20/30	20/400	15	90	Contraction	90
2 cc. 8%	20/25	20/200	10	120	Contraction	90
2 cc. 8%	20/15	20/30	7	60	Normal	50
2 cc. 8%	20/25	HM*	13	138+	Contraction	90
2 cc. 8%	20/30	HM	7	90	Contraction	60
2 cc. 8%	20/20—	20/200	10	60	Contraction	70

* Ability to see hand movements.

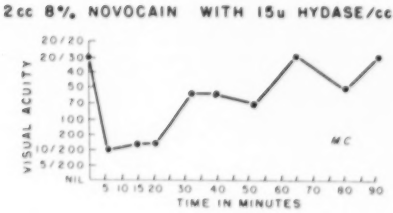

FIGURE 4.

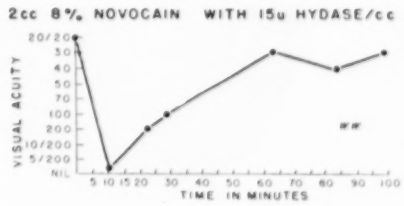

FIGURE 5.

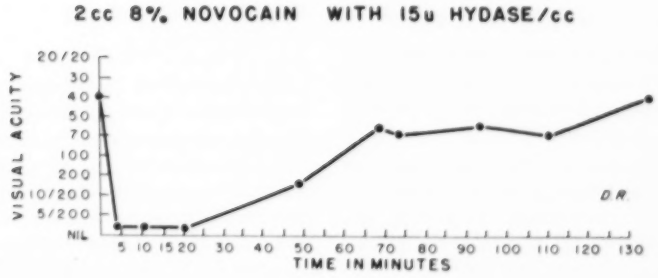

FIGURE 6.

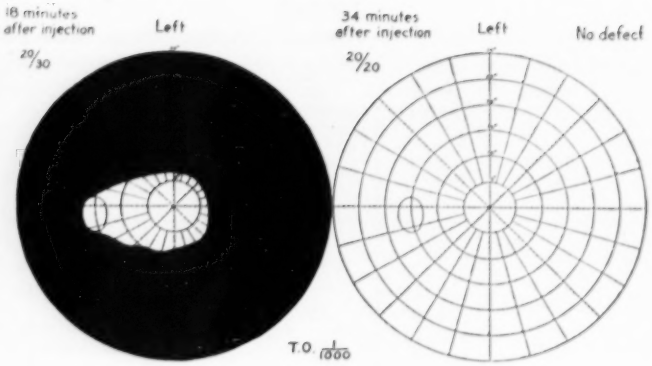

FIGURE 7.

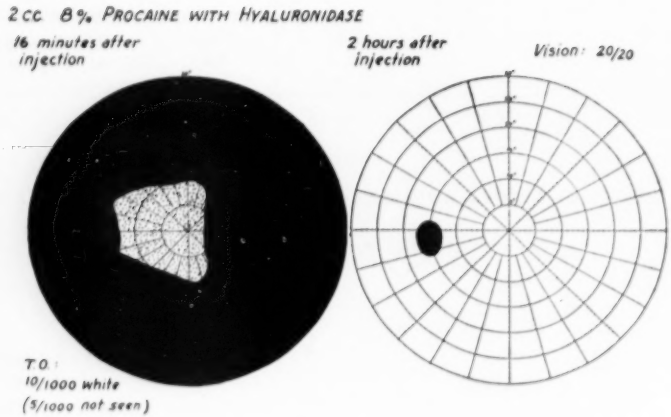

FIGURE 8.

In no case were any changes observed in the appearance of the optic disc or of other parts of the fundus, regardless of the concentration of procaine.

The duration of the impairment of optic nerve function varied from half an hour to two hours. As a rule, the greater the loss of function, the longer its duration. All patients recovered their original vision within two hours.

DISCUSSION

In his excellent work on neuro-ophthalmology, Walsh[7] mentions "transitory loss of vision seen in persons subjected to novocaine injection for the removal of upper teeth or to operations on the orbit." In 1951, Gifford[3] recognized that after the retrobulbar use of xylocaine, a temporary drop in vision, or total loss of vision, sometimes occurred.

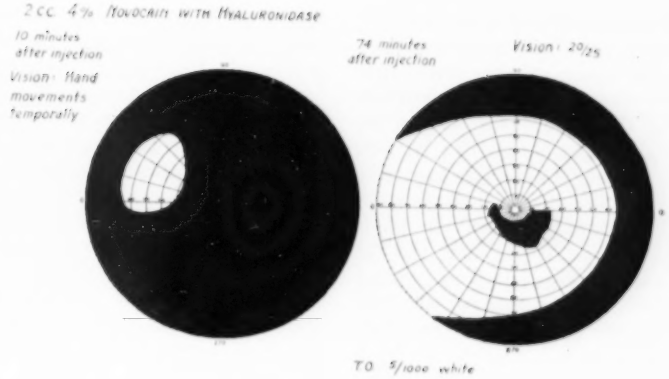

FIGURE 9.

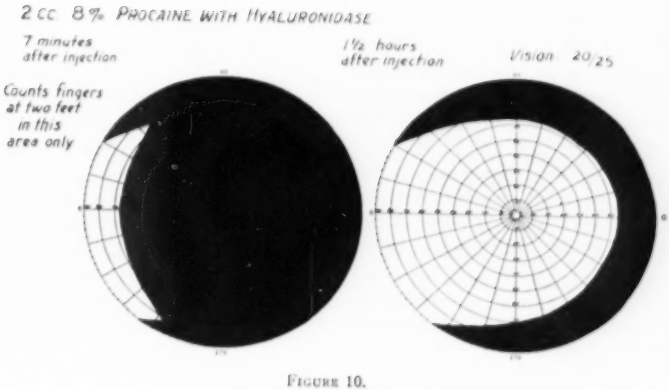
2 cc. 8% Procaine with Hyaluronidase
7 minutes after injection
Counts fingers at two feet in this area only
1½ hours after injection
Vision 20/25

FIGURE 10.

He was probably the first to realize the profound, though transient, effect on the optic nerve of a local anesthetic injected into the orbit. In an article published in July 1954, Russell and Guyton[4] mention temporary anesthesia of the optic nerve following retrobulbar injection of local anesthetics. All of their patients were observed at the time of operation after receiving nembutal and seconal, but the authors felt that the use of 4 per cent procaine had resulted in a transient loss of nearly all light perception in approximately 7 per cent of their patients and that this was increased to approximately 15 per cent when wydase was added to the 4 per cent procaine. When 4 per cent xylocaine plus wydase (5 to 15 u/cc.) was used, approximately 75 per cent of their patients had a transient loss of nearly all light perception.

When a retrobulbar injection of procaine is given according to the commonly practiced technique described in this report, certain well-known events take place.[6] Even before the 35-mm. needle can be withdrawn, the pupil, unless it is under the influence of a miotic, fre-

quently becomes dilated and immobilized.[5] Within a few minutes a partial or complete paralysis of the extraocular and levator muscles occurs.[2] Anesthesia develops in the supraorbital area on the injected side up to the midline, as well as in the cornea, conjunctiva and iris. Gifford[1] rates this motor block as complete if all extraocular muscles are paralyzed. He allows 20 per cent for the block of each of the four recti muscles and 10 per cent for each of the two oblique muscles. According to this grading system, a 30 per cent motor block developed in a patient who received only 2 cc. of saline retrobulbarly. With 2 per cent procaine and hyaluronidase, the block averaged 60 per cent, and with 8 per cent procaine it averaged 84 per cent. Although there were a few exceptions, in general the higher the concentration of procaine, the more effective the motor block and the greater the effect on the optic nerve. In one case the needle was apparently misdirected, since despite the injection of 2 per cent procaine without hyaluronidase, no muscle weakness developed and there was no decrease in vision. Every other patient who received procaine showed partial or complete paralysis of the extraocular muscles and a decrease in vision.

Since no solution can affect the optic nerve unless it is absorbed by that nerve, the procaine must be absorbed by the optic nerve within a few minutes of the injection. The question arises, is there ever any permanent impairment of function due to procaine? We think not, although the following unexplained sequelae should be borne in mind: Several years ago one of us (F.D.C.) had a patient with an immature cataract, normal visual fields for large test objects, and an apparently normal disc preoperatively. This patient developed complete optic atrophy after what seemed to be an uneventful cataract extraction and convalescence. Several similar cases occurring in various parts of the country have come to our attention. The 35-mm. needle, not the long 50-mm. one, was used in all of these cases, and the concentration of novocain was either 2 or 4 per cent. Some observers have felt that trauma to the optic nerve by the needle was the cause of this very rare occurrence. Those who employ retrobulbar injections of alcohol behind eyes that have useful vision should consider whether alcohol, like procaine, may be absorbed by the optic nerve. Optic atrophy is known to have occurred after retrobulbar injection of alcohol.

The reason why a patient with papillitis or retrobulbar neuritis suffers impaired visual acuity and visual changes is merely that a partial or complete sensory block of the optic nerve has developed as a result of the disease process. We now know that a similar temporary block can be produced at will. All of us who have used retrobulbar injections of procaine for many years have no doubt blocked the optic nerve, at least in part, hundreds or thousands of times without realizing it. The practical value during cataract extraction of the resultant transitory loss or diminution of vision has certainly not been generally recognized. When the lens is removed and the bright operating light strikes the eye, the patient not infrequently tries to contract the extraocular and orbicularis muscles in spite of any motor block, and if the block is not efficient, loss of vitreous may occur. If the light is poorly seen, however, or not seen at all, the patient has no such tendency.

SUMMARY

In the course of a study on the effect of the retrobulbar injection of procaine on eyes with glaucoma or suspected glaucoma, the patients' visual acuities were observed and their visual fields plotted. Within a few minutes after the injection, which was performed according to

a technique commonly practiced by surgeons, there was a partial or complete motor block of the extraocular muscles, and a partial or complete block of the orbital sensory nerves, including the optic nerve. The decrease in vision varied from slight (20/20 to 20/30) to profound (20/20 to nil) and tended to be greater the more efficient the motor block. The visual acuity of 10 of a series of 12 patients receiving 8 per cent procaine dropped to 20/200 or less. The value of such a drop in vision during cataract extraction is suggested. Vision in all cases returned to normal in from 30 to 120 minutes and no harmful effects were observed.

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DISCUSSION

HAROLD GIFFORD, M.D., Omaha, Nebraska: I appreciate the opportunity to discuss this paper. The fact that there may be a profound loss of vision when strong solutions of procaine are injected in the orbit will not come to you with quite as great a shock as it did to me the first time I noticed it. It is very reassuring to know that there is no permanent visual loss, even though the vision has been reduced to no light perception. This point has worried me slightly ever since I realized that the solution did penetrate to the optic nerve. On going over my records, I also could find no evidence of permanent loss of vision due to the orbital injection of anesthetics.

For this discussion I have reviewed 371 cases of retrobulbar injections using slightly less than 2 cc. of 2 per cent xylocaine. A 50-mm. long blunt needle was used for most of the injections. Since a blunt needle was used, I am sure the optic nerve sheath could not be penetrated. In this series, no general toxic effect was noted from the xylocaine. There was only an occasional local orbital reaction which was no more frequent than when 4 per cent procaine was used. I am sure that the orbital reaction was less than with the 8 per cent procaine, or with intracaine, or with the combination of pontocaine and novocain solution which I had used previously. In this group of 371 injections, there were 179 intracapsular operations. In only 40 of these cases was the vision tested on the operating table after the operation. Of these 40 cases there were only 21, about 50 per cent, which showed measurable visual loss. Thirteen of these cases showed no light perception. In 6 the vision was reduced to shadows. In 2 there was loss in the nasal field, although the patients were able to count fingers.

In reviewing Doctor Carroll's tables it is quite evident that as the strength of the solution is increased, the data become more consistent and the amount of visual loss has a more direct correlation with the length of duration of the effect. This same tendency was evident when the amount of motor block was checked. The stronger the solution, the greater the amount of motor block. I found the same effect when comparing 2 per cent with 4 per cent procaine. Two per cent xylocaine produced a greater effect than 4 per cent procaine.

The variability in the effect is quite striking. I believe this can be explained chiefly by the anatomy of the orbit. Although the average orbital depth from the infraorbital rim to the optic foramen is 5 cm., the 50 orbits which I measured several years ago showed a variation

between 4.5 cm. and 5.8 cm. In the larger orbits there would be less chance for the solution to penetrate the nerves. In the smaller orbits there would be a greater chance for it to penetrate these structures. Orbital fat may also be a cause for variability; both the amount and the type of fat may be factors. In persons with fat faces and full orbits, it is more difficult to obtain a good motor block. The fat either absorbs the anesthetic or prevents it from reaching the nerves.

The two cases of Doctor Carroll's which showed paralysis of the extraocular muscles and ptosis when injected with saline are not easily explained, but it is possible that some of the procaine may have remained in the syringes or that the actual pressure of the solution injected close to one of these nerves may have produced a transient block of the nerve impulses. The one case that developed optic atrophy must have developed an arterial closure of the nutrient vessels to the nerve either from the adrenalin or from trauma.

Personally, I do not use hydase with my injections, because I believe a prolonged duration of the anesthesia is important. It seems to me that the hydase produces a two-way effect. If the solution is more easily absorbed by the nerve, the spreading factor will also remove it from the nerve more quickly. This did occur in a few cases in which I used hydase. Dr. Guyton brought this out in his paper published this summer.

The type of field loss produced by these injections would indicate that the neuroanatomists are correct in locating the macular bundle deep in the nerve posteriorly. Since there are only four field charts with this paper, this aspect cannot be discussed critically. Perhaps Doctor Carroll or Doctor de Roetth can tell us whether there was a tendency to macular sparing and whether the effect was evident first in the nasal field, as would be expected if the solution were absorbed through the nerve sheath on the temporal side, where it is supposed to be deposited. I have seen a few cases where the nasal field was gone while the temporal field remained intact.

The rapidity with which the optic nerve is influenced by the injection would tend to clear up another debatable anatomical point. Does the cerebrospinal fluid actually communicate with the optic nerve in the orbit? If there were spinal fluid circulating around the optic nerve, this would tend to prevent blocking of the sensory function by dilution of the anesthetic. A neurosurgeon with whom I discussed this problem asked the question, "Has anybody seen spinal fluid leaking from the optic nerve following enucleation?" I personally have not looked for this particular phenomenon, and as far as I know, no spinal punctures have been done following enucleation to determine whether the cerebrospinal pressure is lowered. If there is any real doubt about direct communication, this might be a rather conclusive test to prove or disprove this point.

From a practical standpoint, I am sure that we are indebted to Doctor Carroll and Doctor de Roetth for bringing this dramatic effect of retrobulbar injection to our attention. When profound visual loss is discovered by the surgeon, he may now reassure the patient that this was part of the technique. His voice will now have the proper assurance that the loss of light perception is temporary and that when the dressing is taken off the following day, the patient will be able to see again.

In discussing the use of retrobulbar injections in cataract surgery, I would like to make two points which are not discussed in the paper but which I think important.

First: The production of good motor block and good facial block will reduce the amount of vitreous loss to near the vanishing point. My records show that in the last 179 cases, using the long needle and 2 per cent xylocaine, there was vitreous loss in only 3 (1.6 per cent). In one of these no motor block was obtained. The injection must have been outside the cone. The second vitreous loss was produced when I accidently pulled on the superior rectus suture. The third occurred for no good reason that I could find. There was no pressure on the speculum. The facial block was good. The motor block was 85 per cent. Perhaps this was just not good enough.

The other point concerns the occasional development of an orbital hemorrhage during injection. For the last three years I have used a dull needle. This is obtained by honing off the long bevel, producing a semisharp point with the opening slightly to one side of the end of the needle. I have done 300 injections with the dull needle, and no orbital hemorrhage has occurred. With the sharp needle, either long or short, the incidence of hemorrhage in the orbit was about 1 in 100. In my last series of 100 cases using a sharp needle, it occurred five times. This is why I changed to a blunt needle. It is not always possible to push a blunt needle through the intramuscular septum without using considerable pressure, but by having the patient roll the eye up and in, and by going through the septum as far posteriorly as possible, where the septum is thinner, it can usually be accomplished. In most cases the needle goes in with surprising ease. If it does not pass the septum with relative ease, a sharp needle should be substituted. It

is possible to use the dull needle in at least 95 per cent of the cases. Personally, I would rather use a long blunt needle because it places the solution farther back in the orbit nearer to the motor nerves, where weaker solutions will produce a greater and more constant effect.

DANIEL SNYDACKER, M.D., Chicago, Ill.: It is a great pleasure to discuss this very interesting paper. I think the authors have made a real contribution.

It is remarkable that in spite of the fact that the use of retrobulbar anesthesia is almost universal among ophthalmic surgeons, there have been relatively few controlled studies of the effect produced and there are widespread misconceptions concerning its use. Only recently has it been realized that the phenomena following retrobulbar injection of novocain are entirely ascribable to the nerve blocking action of the anesthetic.

The addition of such substances as adrenalin and hyaluronidase to the novocain enhances the anesthetic effect, but these substances by themselves have no anesthetic action. The anesthesia produced is dependent on those nerves which are blocked and the extent to which they are blocked. In most cases reported by Doctor Carroll and Doctor de Roetth in which there was marked or complete loss of vision, there was marked or complete blocking of the optic nerve; while in those cases in which there was only partial loss of vision, the nerve block was of lesser degree. It might be deducted that if the anesthetic agent was delivered to just the right spot, the blocking was complete. If it was delivered less accurately, the blocking was only partially complete. Certainly in those cases in which there was a central or peripheral visual field loss, this is the logical explanation.

There were a few patients in Doctor Carroll's series with reduction of central visual acuity only, in which he reported a normal field. It is difficult to account for those cases in which there was only this slight blurring of vision. I should like to offer an explanation for at least some of these cases by referring to my own experience. A few years ago while observing the effect of retrobulbar injection of various substances on a series of patients, I subjected myself to a retrobulbar injection of procaine at the hands of one of my colleagues. I might say that it is not an entirely pleasant experience, and I think it might be even a little less pleasant with the dull needle which Dr. Gifford describes. The almost immediate effect coincident with dilatation of my pupil was a blurring of my vision from 20/20 to 20/40 — in that eye. At the same time there was a recession of my near point from 28 centimeters to 50 or 55 centimeters. I assumed that the blurring of vision was due to the unmasking of my latent hyperopia and the dilatation of my pupil as the result of ciliary paralysis. I wonder if Doctor Carroll feels that the blurring in some of his cases might be accounted for by the same mechanism.

DOCTOR DE ROETTH: We would like to thank Doctor Gifford and Doctor Snydacker for their comments and discussion.

As far as Doctor Gifford's question is concerned, we never noticed a central scotoma. Using Traquair's expression, "the whole island of vision was depressed in a sea of darkness and no central scotoma was seen."

I would like to congratulate Doctor Snydacker for his truly scientific zeal. We certainly did not subject ourselves to a retrobulbar block.

It is possible that there was some depression in accommodation in those few cases where the visual decrease was only minimal. However, in most of our cases the visual depression was so marked that even if there were some loss of accommodation, one could not measure it.

EXTENDED RETROHYOID RADICAL NECK DISSECTION FOR MALIGNANT TUMORS OF THE ORAL CAVITY AND NECK

JOEL J. PRESSMAN, M.D.
LOS ANGELES, CALIFORNIA

RECENT reports concerning the results of treatment of tumors which metastasize to the lymph nodes of the neck have been most satisfying. Since the introduction of newer surgical techniques,[7,19,24,27,39,43] expectancy for cure has risen from essentially zero to about 30 per cent. Reflecting upon the possibilities for still further improvement, one's attention is directed to the area adjacent to the midline of the neck both above and below the hyoid bone, a region which has not always been given the surgical consideration it deserves.

Justification for enlarging the scope of radical neck dissection to include routinely more of the structures adjacent to the midline is based upon biologic, anatomic, clinical and pathologic evidence. This holds true whether the operation is performed in continuity with the structures of the oral cavity or as an independent isolated procedure.

BIOLOGICAL CONSIDERATIONS

Carcinomas of the head and neck, like other cancers, spread by way of the lymphatics and blood stream[17] and by direct extension. Of special interest is the observation that the entrance of tumor cells into the lymphatics or blood stream is influenced by physical factors such as pressure. Manometric studies verify the fact that increases of pressure within the substance of the tumor enhance the likelihood of cells entering either the vascular or lymphatic channels;[47] thus, anything which increases pressure within the tissue favors embolization of tumor cells. No tumors of the body are more likely to be exposed constantly to increase in pressure than those of the mouth and pharynx, where the acts of mastication, deglutition, speech and closure of the jaw, sometimes with great force even in repose, enormously increase the pressures concerned. It is understandable, therefore, that spread of these tumors to neighboring lymph nodes occurs frequently.[10] It is likely to occur early in the history of the disease, the tumor oftentimes having metastasized throughout the lymphatic system before involvement of the nodes is clinically recognizable.

The spread of intraoral cancer into the lymphatic system is, for the most part, by predictable channels. A lesion located to one side of the midline almost invariably metastasizes first to the nodes on the same side. Many of the tumors, however, eventually metastasize to the opposite side,[6] a fact which the surgeon should bear in mind, since it is reasonable to suppose that this incidence can be lessened by meticulously removing whatever resectable lymphatics exist in and about the midline of the neck, across which the metastatic cells must pass.

From the Department of Surgery, Division of Head, Neck and Plastic Surgery, University of California Medical Center, Los Angeles.

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Spread beyond the cervical lymphatics is not frequent, so that death from cancer of the head and neck ordinarily occurs with the lesion still confined above the clavicles,[10,24,29,35] patients succumbing to inanition, hemorrhage, respiratory obstruction or infection.[32] Estimates as high as 80 per cent[2,45] are often given for the incidence of lesions remaining confined to the limits of the head and neck and therefore surgically accessible. The fact that reported cures resulting from present standard operative procedures are still infrequent despite this localization to the neck is adequate evidence, on the basis of biology alone, that the rational approach to the solution of the problem will not spare local tissue by limiting the field of the operation. On the contrary, biological considerations indicate that as much of the neck should be resected as possible, since if we can surgically circumvent the local spread, we can in most cases cure the patient.

RATIONALE FOR EXTENDING THE FIELD OF RADICAL NECK DISSECTION UPON THE BASIS OF THE ANATOMY OF THE LYMPHATICS OF THE NECK

Radical neck dissection, either in continuity with structures of the oral cavity or as an independent procedure, must, therefore, follow a pattern dictated by studies upon the area of local recurrences, an area which is determined by the anatomy of the lymphatics of the neck.[32] Since it is generally agreed that a proper radical neck dissection should remove the lymphatics from the mandible to the clavicle and from the anterior edge of the trapezius to the midline of the neck,[14,19,20,27,38,43] in reviewing the anatomy of these lymph nodes and channels, special attention will be directed to those underlying the ribbon muscles above and below the thyroid cartilage, as well as those in the midline above the hyoid bone, since these are the most frequently neglected.

Many of these lymphatics underlie not only the midline but also the thyroid gland, so that resection of a lateral half of this structure should be included. Why the lymphatics of the middle region of the neck should be considered immune to the spread of cancer and often overlooked in neck dissections is one of surgery's great mysteries. Since this omission is such an obvious surgical oversight, the region above the hyoid bone, representing the site of the suprahyoid muscles, combined with the region below the hyoid bone from the midline to the lateral border of the ribbon muscles and including the thyroid gland, will be referred to collectively as "the forgotten zone." Besides muscles and the thyroid gland it contains arteries, veins, and numerous lymphatics.

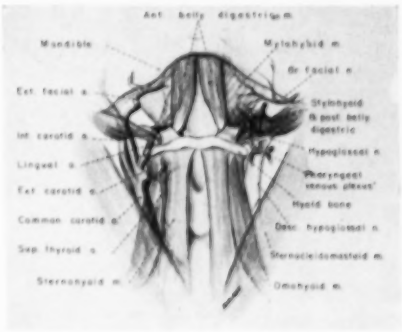

FIG. 1—The "forgotten zone" of neck dissection.

The muscles of the forgotten zone comprise two groups: the first, below the hyoid bone (fig. 1), includes the sternohyoid, sternothyroid, thyrohyoid and omohyoid muscles; the second, above it, includes the mylohyoid, hyoglossus, geniohyoid, stylohyoid and digastric muscles. It is surprising to note that 22 muscles attach to the hyoid bone. Resection of all eleven muscles on the involved side is important from two standpoints: first, their sheaths, as in all cancer-bear-

ing areas, are richly supplied with lymphatic capillaries, so that the muscles themselves offer a site of recurrence; second, lymphatic pathways representing potential routes for the dissemination of cancer lie under them. These pathways consist of lymphatic channels which, although not well defined, follow the distribution of the deeply placed blood vessels and even greatly exceed the latter in number (fig. 2).[16]

Not only are lymphatic channels found in the sheaths of the muscles but their rich network follows the course of the blood vessels, making the vascular distribution under this muscular bed particularly important. The deep vessels of the midline area below the hyoid bone rise from the external carotid artery and from the thyrocervical trunk. Several of the branches travel horizontally to the midline and anastomose across it with corresponding vessels of the opposite side. Since these vessels are accompanied by lymphatics which also cross the midline, it is evident that their anatomic bed presents a potentially important route for the passage of carcinomatous rests from one side of the neck to the other.

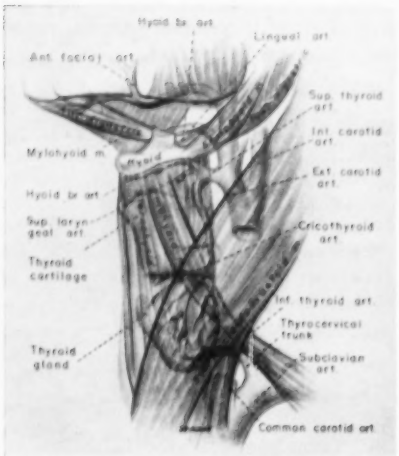

FIG. 2—Vascular distribution of the forgotten zone. The vessels are accompanied by lymphatics.

The superior thyroid artery, a branch of the external carotid, passes downward beneath the omohyoid, sternohyoid and sternothyroid muscles, and in its downward course its branches extend to these and other ribbon muscles. A hyoid branch roughly parallels the hyoid bone along its lower border. This latter vessel is one which freely anastomoses with its fellow of the opposite side. The superior laryngeal vessels are similarly deeply placed and, with accompanying lymphatics, pass through the thyroid membrane deep to the thyrohyoid muscle to enter the interior of the larynx. Still lower, the superior thyroid artery gives off a cricothyroid branch between the thyroid and cricoid cartilage, and it also freely anastomoses with the corresponding vessel of the opposite side. Branches of the vessels to the thyroid gland are numerous and freely anastomose with branches of the inferior thyroid artery, which are also located deep to the thyrohyoid and sternothyroid muscles. These are accompanied by the lymphatic drainage of portions of the thyroid gland itself.

Above the hyoid bone, the lingual artery and its accompanying lymphatics pass beneath the hyoglossus, between it and the genioglossus.[16] Therefore, in order to expose and remove the lymphatics accompanying the blood vessels above the hyoid bone, it is necessary to resect the digastric, stylohyoid, mylohyoid and hyoglossus muscles. Any lesser dissection of this region fails in its intended purpose.

As evidence of the anatomic importance of this route of spread, it is to be noted that all the vessels enumerated, except the inferior thyroid, are immediate or secondary branches of the external carotid artery, which is intimately related

anatomically to the deep cervical lymphatic chain. Into this lymphatic chain empty practically all the lymphatics of the head and neck, including those of the skin, mouth, pharynx, tongue, jaws, and the salivary and thyroid glands. Since this deep cervical (or internal jugular) chain is the principal lymphatic route in the neck involved in the dissemination of cancer, not only the area which it traverses should be resected but also that which it drains. Since much of the area which it drains lies deep to the muscles attached to the hyoid bone, the removal of both areas becomes an absolute necessity. A detailed description of the distribution of the lymphatic glands and lymphatic channels of the neck, from which figure 3 was adapted, may be found in Rouviere's text (fig. 3).[33]

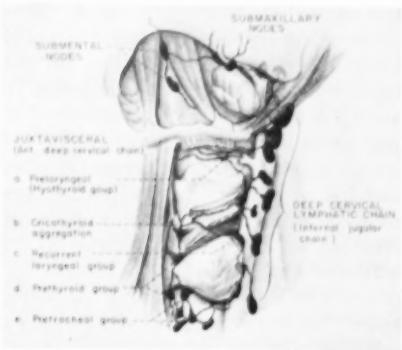

FIG. 3—Lymphatic bed of the forgotten zone. (After Rouviere)

Apart from these lymphatic channels, the lymphatic nodes of the "forgotten area" can, for purposes of description, be conveniently divided into two categories: those above and those below the hyoid bone. The latter comprise the previously mentioned anterior cervical chain, which is subdivided into two principal groups: the more superficial anterior jugular chain, and the deeper juxtavisceral chain. The anterior jugular chain (not illustrated) consists of a few small nodes limited in front by the anterior layer of the deep cervical fascia. These form a constant lymphatic pathway, which Rouviere has been able to inject. It drains the skin and muscles of the anterior region of the neck. The juxtavisceral nodes (deep cervical nodes of Bartels or the anterior deep cervical chain of Poirier and Cuneo) are placed more deeply and are composed of four subgroups: (1) prelaryngeal, (2) prethyroid, (3) pretracheal, and (4) laterotracheal.

The prelaryngeal group seems to be inconsistent except for an intercricothyroid aggregation (cricoid nodes) found on the cricothyroid membrane. Such nodes were found in nearly half the specimens examined. The prethyroid group is not constant, but in certain instances nodes are found near the superior and inferior margins of the thyroid isthmus. The pretracheal group is constant and is placed either in or superficial to the pretracheal fascia on the front of the trachea, and lateral to it along the recurrent laryngeal nerves.

These subgroups constituting the juxtavisceral division of the anterior cervical chain form a pathway of nodes beneath the ribbon muscles and extend from just below the hyoid bone to the clavicles. The lymph channels constituting this system follow the blood vessels, as previously described, and empty into the deep cervical (internal jugular) chain.

The nodes above the hyoid bone lie chiefly in the submental area, in the region bounded by the mandible above, the hyoid bone below, and the anterior belly of the digastric muscles laterally. They may lie directly on the mylohyoid and digastric muscles deep to the fascia or in the subcutaneous tissues over them and are really part of the anterior jugular chain. On occasion, nodes are found

immediately above the hyoid bone and may be placed medially or laterally or both. The efferent vessels of these nodes drain directly into the submaxillary group and thence into the internal jugular chain. Extension of malignant lesions may occur in a retrograde manner so that submental nodes might be involved in any malignant process involving either the submaxillary or internal jugular chain. It is logical, therefore, to dissect these nodes routinely in all radical neck dissections rather than solely in the resection of midline tumors.

From the floor of the mouth, lymphatic channels pass along the course of the lingual artery and its branches so that at least part of the lymphatic system of the area is located deep to the mylohyoid, hyoglossus, geniohyoid, digastric, and stylohyoid muscles. These regions, too, may be involved not only by direct extension but in retrograde fashion from the submaxillary chain or the frequently involved internal jugular chain.

Because lymphatics are found not only in well defined channels but in the muscle sheaths and along blood vessels as well, it appears shortsighted indeed to attempt any partial resection of a given area. Whether the lymphatic vessels are afferent or efferent is of no very great consequence since, as Martin[27] has stressed:

> The lymphatic spread of cancer, although at first orderly, proceeding from the source of origin to a regional lymph node, eventually proceeds in a less orderly manner and extends through the lymphatics even in a retrograde fashion.

Rouviere[33] substantiates this by stating that due to dilatation of the lymph vessels from obstruction, cancer may progress along the lymph stream in a direction opposite to its normal flow. Ward and Hendrick[43] lend further evidence by pointing out that retrolymphatic and cross metastases occur, and Huffman and Lierle[19] stress the fact that blockage of the lymphatic stream at any one point by metastatic cancer can reroute the lymphatic flow in any other direction. Therefore, in all radical neck dissections, even for lesions arising in sites which do not directly drain into the glands of the forgotten area, the removal of the lymphatics of this region is indicated whether their communication with the lesion is by afferent or efferent vessels.

CLINICAL AND PATHOLOGIC EVIDENCE JUSTIFYING AN EXTENDED RADICAL NECK DISSECTION

A proposal for extension of the surgical field to include routinely more of the structures about the midline is considered justified not only on the grounds of anatomy and the biology of these tumors but from clinical evidence as well. Local recurrences following routine radical neck dissection are common. Most of them, it is true, are found laterally along the deep cervical chain, but the midline area is by no means immune. In an examination of 33 specimens displaying extension of carcinoma into the lymph nodes from oral lesions, Royster and his co-workers[34] found an average of 8 involved nodes in the suprahyoid area and an average of 36 nodes in the infrahyoid area, which is in distinct opposition to any concept citing the infrequency of involvement of the forgotten zone. Martin and his associates[27] report a 20 per cent recurrence rate of metastasis to the opposite side of the neck, probably by crossing the midline. Martin further reports a 23.2 per cent recurrence "just outside the operative field." Since this undoubtedly represents the common experience and includes midline structures, is it not in itself sufficient evidence to justify extending the operative field in this direction?

Whether an extended retrohyoid radical neck dissection, as described, will appreciably lessen this incidence is a mat-

ter of conjecture at this time, although on theoretical grounds, and until evidence is presented to the contrary, it would seem that the surgical principle involved should stand, since it not only extends the surgical field of operation to the midline but fulfills Huffman and Lierle's requirement, "It must be our aim to remove *in toto* the entire lymph drainage area containing the metastases, plus at least the lymphatic tissue next beyond it."[19] The lymphatics of the forgotten zone are next beyond the all important deep cervical chain.

Further strong clinical and pathologic evidence for routinely including all the suprahyoid and infrahyoid muscles and the thyroid hemigland is the fact that it is not possible preoperatively, or even by evidence obtained on the operating table, to rule out the presence of metastatic glands in the neck, which, of course, includes the midline region, an area particularly deceiving because of overlying muscles and the thyroid gland. This point is clearly brought out by Tailheffer[42] and is further discussed under "Indications."

INDICATIONS AND CONTRAINDICATIONS

In a significant percentage of cases there is no histologic evidence of metastatic cancer in nodes palpable preoperatively.[2] On the other hand, throughout the literature a high incidence of metastatic cancer is recorded in cervical lymph nodes as not palpable preoperatively but as present in the nodes of specimens removed "prophylactically." This incidence is cited, for example, by Tailheffer[42] of the Curie Institute, who reports a series of 244 cases of cancer of the tongue in which 43 per cent had positive nodes not palpable prior to surgery but discovered either at the time of operation or subsequently by the pathologist. Many other reports firmly support Tailheffer's findings.[2,3,7,10,34,39]

Indications for routine radical neck dissection and justification for automatically extending it to include more of the midline structures must, therefore, be considered with regard for the facts that one is not able to rule out the presence of involved nodes by palpating the neck and that even when they are clinically demonstrable, it is impossible to determine the number, distribution and sites of the involved nodes. For these reasons, performing what is generally known as a prophylactic neck dissection is not entirely prophylactic at all in the sense that it is intended solely to prevent the future dissemination of cancer. Its performance represents, instead, the recognition of factors concerning the biology of these tumors and the likelihood of their early local dissemination without clinical evidence of this spread. It is, therefore, an acknowledgment of the fact that one is not able to establish the presence or absence of involvement of the lymph nodes of the neck. Since metastases to the lymph nodes of the neck occur early, the conservative point of view would be to assume that upon recognition of the disease of the primary site, involvement of the cervical nodes on the homolateral side has already taken place or is imminent and that radical neck dissection is indicated as part of the primary procedure. This assumption applies especially to tumors of the tongue, pharynx, tonsillar area, floor of the mouth and gingiva,[6] it being generally agreed, however, that certain early tumors of the lip and basal cell lesions are exceptions. It is taken for granted that resection of the primary site and radical neck dissection are to be combined and performed in continuity. It is generally agreed that the extension of malignant disease from the head and neck into the cervical lymph nodes, or the threat of such extension, is an indication for radical neck dissection, provided the primary lesion can be controlled;[8,10,19,38] however, the criteria

used to determine whether extension has taken place or is about to take place, and varying opinions as to the controllability of any particular primary lesion, constitute sources of disagreement. The absence of palpable lymph nodes in the neck is certainly no guarantee that metastasis into them has not taken place.

Slaughter, Roeser and Smejkal[35] have divided indications for the combined commando procedures, including radical neck dissection, into two categories, relative and absolute. Absolute indications consist of clinical situations in which squamous cell carcinoma of the buccal mucosa, gingiva, or floor of the mouth, or adenocarcinoma or mucoepidermoid carcinoma of the salivary glands invades the mandible by direct extension and produces cervical node metastases. This concept includes (1) carcinomas of the anterior palatal area, tonsil and adjacent pharyngeal wall, (2) cancer either in or about the mandible which has persisted after radiation, and (3) extensive tongue lesions which have recurred after radiation, whether or not the mandible seems to be involved. The mandible is resected because its presence prevents adequate surgical approach to adjacent tissues.

Relative indications comprise those situations in which the carcinoma is not far advanced but still not radiocurable. These authors[35] believe that studies of large series of borderline cases will be required for the proper evaluation of relative indications in order that the extended commando technique may be properly applied to specific earlier cases in the hope of improving the 25 per cent cure rate. Recalling the high incidence of clinically silent extensions from primary sites to cervical glands, it seems that in time a larger group of cases now considered as relative will become absolute. This trend is already unmistakable.

Contraindications to surgery must be considered, bearing in mind the fact that untreated cancer is invariably fatal and that very little can be offered by other methods. Insofar as the primary lesion is concerned, radiation in some instances offers an alternate but, as pointed out by Huffman and Lierle,[19] universal opinion holds that regardless of whether radiation or surgery is chosen for the primary lesion, metastases to the neck must be treated surgically; the invoking of contraindications therefore sentences the patient to certain death.

Only two absolute preoperative contraindications appear to be acceptable, the first being evidence of distant metastases and the second an uncontrollable primary lesion. Arguments citing as contraindications fixation to surrounding tissues and involvement of vital structures are valid only if the surgeon has opened the neck and has found that he cannot remove the metastatic tumor. To assume preoperatively that this is so, considering that life is at stake, is an error, for one is often fooled, and seemingly impossible dissections are often the easiest to perform. Neither should one accept as a contraindication the biologic character of the tumor—its speed of growth, invasion qualities, degree of differentiation of the cells, Broders' classification, or invasion of the capsule of glands, which is often incorrectly assumed to be associated with a poor prognosis.[34] In no way can the probability of distant metastases or curability be determined with certainty by the morphology of the tumor.[5]

It is not always good policy to avoid surgery of this character for the sole reason that the medical status of the patient seems to indicate that the stress of the operation itself cannot be withstood, for neither can the cancer. It is difficult to predict the ability of any patient to withstand surgery, since often those with severe complicating illnesses withstand radical surgery surprisingly well, particularly on peripheral portions of the body.[29] Thus, in keeping with Mar-

tin's philosophy,[27] this is an opportunity to convert the certainty of death to the uncertainty of life, an apparently poor general physical condition of the patient notwithstanding.

TECHNIQUE

Quite apart from the discussion of the relationship of technique to end results, one can recommend the method of hemihyoidectomy with retrohyoid dissection, as it will be described, for the ease with which it can be done. This dissection follows the natural planes of cleavage, much of it along the pharyngeal aponeurosis which forms the floor of the surgical field above the thyroid cartilage.

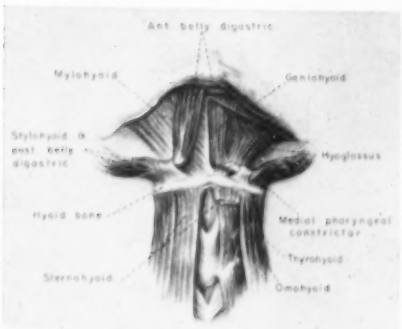

FIG. 4—The hyoid bone is the keystone of neck dissection.

The technique of extended retrohyoid radical neck dissection is based upon the concept that the hyoid bone is the surgical keystone of the neck, since, as I have pointed out, 20 or 22 muscles (depending upon whether or not one considers the chondroglossus as a separate muscle or part of the hyoglossus) are attached to it. These extend upward into the floor of the mouth and tongue, downward to the sternum, on each side of the midline, and laterally to the region of the mastoid (fig. 4). Of all the muscles concerned in radical neck dissection, only the sternocleidomastoids are not so attached. These 20 muscles cover the entire anterior aspect of the neck over a wide arc of its circumference, and all of them, including the ribbon muscles and those of the submental and submaxillary regions, can be resected in a single bloc provided their attachments to the hyoid bone are left undisturbed. The operation can be performed through any of the standard incisions, the author's original preference having been for the arc above and below, joined by a drop line. One patient, however, recently demonstrated atrophy and sloughing of the flaps for reasons which are not clear, and we are seriously considering adopting as routine the incision described by Fraser,[13] consisting of the usual upper limb and a curved S-like vertical limb. In two recent cases this incision has impressed me with its worth from the standpoints of exposure, healing, and functional and cosmetic end-results. The inclusion of the platysma and overlying fascia, together with the most superficial layer of deep fascia, as part of the skin flaps is considered reasonable on the premise that if the superficial lymphatics have been invaded, the skin is probably already involved and should be resected with the platysma.[6,25]

As in the standard procedure, the flaps are dissected free to the level of the mandible above, the clavicle below, the trapezius posteriorly and past the midline of the neck anteriorly, so as to isolate and avoid the mandibular branch of the facial nerve at its lowest point near the facial vessels as they cross the mandible. The dissection, if carried out from anterior to posterior, is begun by severing the sternal and clavicular heads of the sternocleidomastoid (fig. 5), after which we deviate from the standard procedure by severing the sternal attachment of the sternohyoid and the more deeply placed sternothyroid muscles. If, however, it is begun posteriorly, an incision is made through the fat deposit along the margin of the trapezius, the posterior belly of the omohyoid is cut,

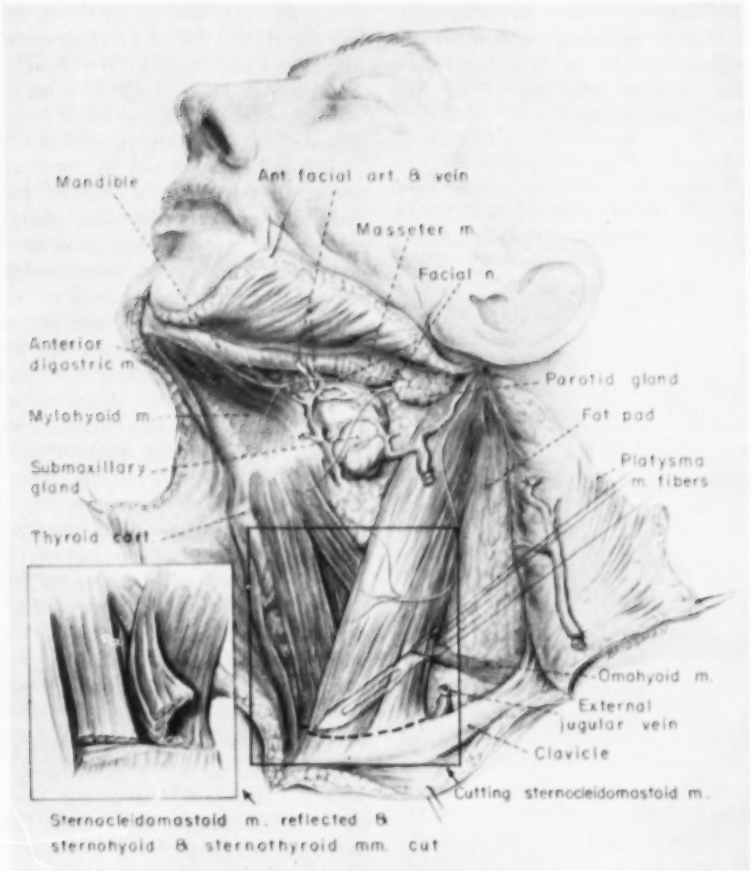

FIG. 5—Skin flaps reflected: sternocleidomastoid, sternohyoid and sternothyroid to be divided.

and the dissection is continued forward on the clavicle to the anterior margin of the sternocleidomastoid, severing its insertion at the clavicle and sternum, as well as those of the two aforementioned ribbon muscles.

The severed end of the sternocleidomastoid, the two severed ribbon muscles and the posterior belly of the omohyoid are encircled by a strong suture-tie to incorporate them into a single bundle for ease of manipulation. These are reflected upward (fig. 6) together with the fat pad of the posterior triangle, thereby exposing the carotid sheath, which is entered for the purpose of tying and cutting the internal jugular vein. Four ties, two above and two below, are ordinarily placed instead of the three demonstrated. The superior segment of the jugular vein is then included as part of the reflected mass.

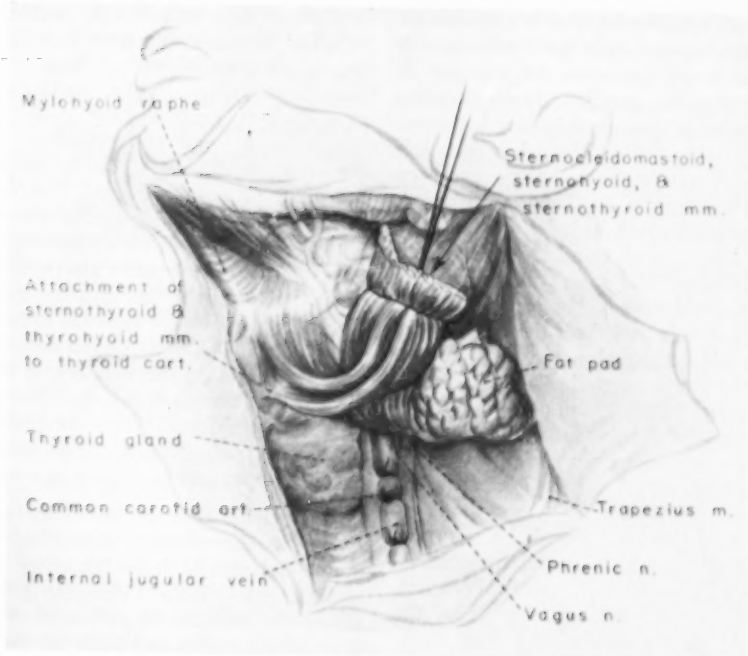

FIG. 6—Development of inferior limit of resected block including infrahyoid ribbon muscle.

The structures being resected are easily separated in a normal plane of cleavage superficial to the third layer of the deep fascia, exposing the trachea, the thyroid gland covered by its capsule, the contents of the carotid sheath and, as the dissection proceeds upward, the cricoid cartilage, the cricothyroid membrane and the cricothyroid muscle. Posteriorly we come upon the phrenic nerve, the cervical trunks and the muscles forming the floor of the posterior triangle (fig. 7). The full length of the spinal accessory nerve is not demonstrated, since it is hidden in the muscle mass being reflected and in the fatty tissues of the posterior triangle.

FIG. 7—Sternothyroid and thyrohyoid dissection in continuity. Hyoid bone about to be split in midline.

The accompanying illustrations do not demonstrate removal of the thyroid hemigland, but I am becoming convinced that radical neck dissection should include hemithyroidectomy in order to remove neighboring deep lymphatic nodes as well as those lymphatic channels accompanying the superior and inferior

thyroid vessels. In the description of this technique, and in the illustrations, it will be noted that while the principle of extending the operation to the midline of the neck has been followed, the region of the thyroid gland has been left intact. Recently the technique has been further extended so that the operation, as now performed, includes an additional step. The thyroid gland is split through the isthmus, and after tying the inferior thyroid vessels, the lobe on the involved side is peeled off the trachea, the recurrent laryngeal nerve having been isolated and preserved. Half of the thyroid gland is, therefore, permitted to remain by the attachment of the superior thyroid vessels as part of the resected mass. This added step is deemed necessary not only on anatomic grounds by reason of the presence of the recurrent laryngeal, prethyroid and pretracheal group of glands, but for clinical reasons, since recurrences in the region of the thyroid gland, particularly deep to it, have been observed. No harm from the lack of thyroid or parathyroid secretions has resulted from this removal.

The resection continues upward until the oblique line of attachment of the sternothyroid muscle to the ala of the thyroid cartilage is reached. The thyrohyoid muscle above shares this attachment to the oblique line, and in order to permit the inclusion of these muscles in continuity, this common fibrous attachment is severed from the thyroid cartilage by using a scalpel blade placed flat to the ala. The dissection can then be continued upward with the two muscles remaining in continuity, separation from the thyrohyoid membrane being readily accomplished as far as the level of the hyoid bone. The external carotid artery is tied above the origin of the superior thyroid artery, and cut. The hyoid bone, which is steadied by grasping it with towel clips placed on either side of the midline, is then sawed through in the midline or split with heavy shears (fig. 7). The lateral half, which is to be removed, is grasped with a hook at the point of severance, and by exerting traction upon it ventrally, the underlying structures are put under tension. The posterior attachment of the hyoid bone can then be easily severed, freeing it from its bed but allowing its muscles, with the exception of the medial pharyngeal constrictor, to remain attached. The dissection continues beneath the geniohyoid and hyoglossus muscles, exposing the wall of the pharynx over a wide area and incorporating the suprahyoid muscle groups (digastric, stylohyoid, mylohoid and geniohyoid) into the resected mass. The medial constrictor is severed from the bone and permitted to remain.

An incision through the raphe anteriorly, in the midline, separating one mylohyoid muscle from the other, discloses a clearly defined cleavage line between the muscles of one side of the floor of the mouth and those of the opposite side. When this has been accomplished, the surgical specimen is easily dissected upward to the level of the mandible. The submaxillary gland is simply lifted along with the rest of the specimen, since its entire bed is included. The anterior facial vein is tied off superficially; and the mylohoid, submental and hypoglossal veins are also tied off when and if they can be identified.

If the facial vessels were not cut and tied when the skin flap was elevated, this is now done, taking care to avoid, whenever possible, the mandibular branch of the facial nerve. If the lingual vessels lying on the geniohyoid muscle deep to the hyoglossus can be brought into view, they are isolated, tied and cut; if not, this is postponed until the dissection has become further advanced.

From this point on, the technique varies depending upon whether only the radical neck dissection is to be per-

formed or the operation is to be extended to include the mandible, floor of the mouth, and structures of the oral cavity. If only the radical neck dissection is to be done, the specimen is dropped back into its general anatomic position, and beginning at the midline (fig. 8), the anterior belly of the digastricus is cut at its attachment to the symphysis of the mandible. The mylohyoid is sectioned as closely as possible to its origin on the mylohyoid line, and the geniohyoid is severed near the symphyis. The fascia binding the superficial aspect of the submaxillary gland is split along its bony attachment.

When these structures have been cut, blunt dissection will expose the hypoglossal nerve lying between the mylohyoid and hyoglossus muscles. It can be dissected free and preserved or, if indications warrant, severed and included in the specimen, the resulting paralysis of the corresponding side of the tongue being entirely tolerable. There is some question whether resection of this nerve should not routinely be done on the same grounds that justify routine resection of the spinal accessory nerve. The submaxillary duct just superior to the hypoglossal nerve is tied and cut. The hyoglossus muscle is sectioned high up under the mandible (higher than demonstrated in the illustration). The dissection is continued laterally following the posterior belly of the digastric muscle and cutting across the lower pole of the parotid gland, which is included in the specimen. The posterior bellies of the digastric and stylohyoid muscles are followed to the region of the mastoid tip (fig. 9), and after retracting these upward, severing them now or later as expediency dictates, the internal jugular vein is once again tied and cut between two ties above and one below. The spinal accessory nerve, if it is not to be preserved, is cut again at this high level, and the specimen is detached by severing the mastoid attachment of the sternocleidomastoid and whatever facial attachments still remain posteriorly. It is to be noted that the suprahyoid muscles have been resected to the level of the genioglossus, which forms the bed of the floor of the mouth (fig. 10). The superficial view of the resected specimen is demonstrated in figure 11, and the deep view in figure 12.

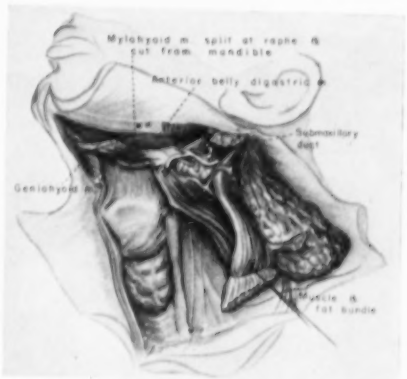
Mylohyoid m. split at raphe & cut from mandible
Anterior belly digastric m.
Submaxillary duct
Geniohyoid m.
Muscle & fat bundle

FIG. 8—Pharyngeal aponeurosis exposed. Mylohyoid raphe split. Suprahyoid structures being separated from mandible.

EXTENDED RETROHYOID RADICAL NECK DISSECTION PERFORMED IN CONTINUITY WITH HEMIMANDIBULECTOMY AND RESECTION OF THE FLOOR OF THE MOUTH

Radical neck dissection performed alone as a discontinuous procedure can, of course, be properly applied only in those cases in which the primary lesion has been adequately controlled. For example Carroll,[6] while not championing it, cites possible appropriate instances for its use as an isolated procedure when a primary lesion is radiosensitive but metastases to the cervical lymphatics are not. He states that there has evolved in

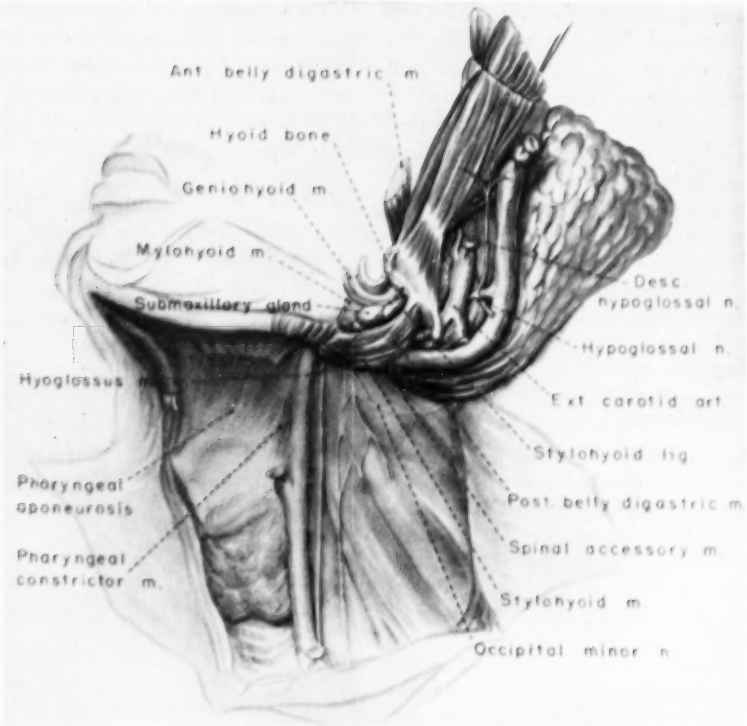

FIG. 9—Specimen immediately prior to removal. Sternocleidomastoid, stylohyoid and posterior belly of digastricus will be severed at superior limit, and internal jugular vein tied near base of skull. Lower pole of parotid gland included in specimen. Hypoglossal nerve sacrificed (optional).

many centers a plan of dissociated therapy in which radical neck dissection is carried out independently of the control of the primary lesion. It is also suitable as the second part of a bilateral neck dissection, either immediate or delayed, and for recurrences in the neck when previous treatment had been directed solely to the local lesion.

It is generally agreed, however, that good cancer surgery requires that the primary lesion, the cervical lymphatics and the intervening structures be resected in continuity. The chief application of extended retrohyoid radical neck dissection is therefore not as an independent isolated procedure but as part of an en bloc dissection carried out in continuity with a primary lesion. The concept of in-continuity dissection is, as Slaughter, Roeser and Smejkal[35] point out, particularly applicable to intraoral cancer. When these authors, for instance, adopted Martin's one-stage commando technique, performing in continuity a radical neck dissection, hemimandibulectomy and resection of involved soft parts, the improvement in their results was striking.

The technical problems inherent in an en bloc procedure, in which resection of the involved oral area, partial mandibulectomy and radical neck dissection are performed in continuity, are actually

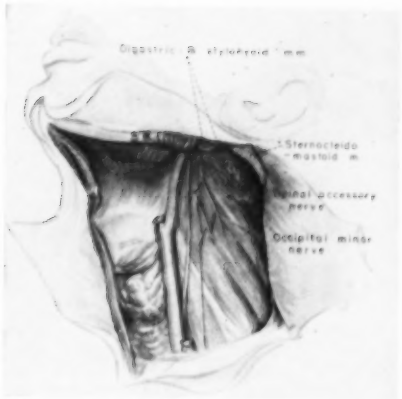

FIG. 10—All nonvital structures (except lobe of thyroid gland) removed from midline to trapezius and from mandible to clavicle. Recent operations have included hemithyroidectomy.

simplified by adapting the principles of the extended retrohyoid approach. The plane of cleavage along which the dissection proceeds is immediately superficial to the pharyngeal aponeurosis, which is the natural anatomic approach to the structures in and about the floor of the mouth.

In the combined procedure, the contents of the neck remain attached to the mandible. The dissection is carried out as described to the point at which the hyoid bone is split. The raphe between the right and left mylohyoid muscles is cut in the midline in order to develop further a line of incision in the cleavage plane between the right and left hyoglossus and geniohyoid muscles. It is noteworthy that a fibrous septum joins the two halves of the tongue, passing downward between the muscles to join the hyoid bone.[16*] After splitting the hyoid bone, it is this fibrous septum which delineates the midline route to be followed through anatomic cleavage planes to the floor of the mouth into the substance of the tongue itself.

If the primary incision has not been made through the lower lip, this is now split through and through in the midline, continuing over the chin and joining the medial limit of the upper arm of the primary incision in the neck. An incision is made in the mucosa of the buccogingival sulcus from the midline as far laterally as possible, after which attention is directed to the interior of the mouth. The mucosa of the floor of the mouth is split

FIG. 11—Superficial surface of specimen; structures including forgotten zone removed en bloc.

FIG. 12—Deep view of resected specimen.

*Gray, p. 1137

just internal to the mandible over an area indicated by the extension of the intraoral resection to be performed. The details of the ordinarily confusing surgical treatment of the intraoral portion of the operation, including surgical repair, are well presented in Morfit's illustrations.[29] The mandible is split lateral to the midline with a Gigli saw in order to preserve the curve of the chin. This site of sectioning lessens to a great extent the postoperative cosmetic and functional defect. In lesions involving the posterior aspects of the oral cavity, disarticulation at the glenoid fossa is desirable, provides an easy closure, and seems to give the least postoperative disability. In Slaughter's experience, however, preserving even part of the ascending ramus allows better secondary restoration of function by bone grafts or prostheses.[35] If the ramus is to be preserved, a Gigli saw cut is made through it at the desired level. Detachment of the hemimandible at the glenoid fossa is greatly simplified by the original Gigli cut through it, since under these conditions it can be retracted downward and outward, bringing the pterygoid muscles, which need to be severed, into view. This puts the ligaments, capsule and attached muscle fibers under tension so that they can be cut as the condyle itself is gradually rendered more mobile by the sectioning of successive structures.

After severing the masseter muscle, the entire mass remains attached only by the structures of the floor of the mouth. Attention is directed from below to the well defined line of cleavage between the genioglossus muscles, which may virtually be separated from each other by blunt dissection (fig. 13). The incision through the tongue itself, delineating the extent of the resection, is made by electrocautery. Even if a small fraction of the tongue is to be resected, this is greatly facilitated by putting the tongue under tension with traction sutures

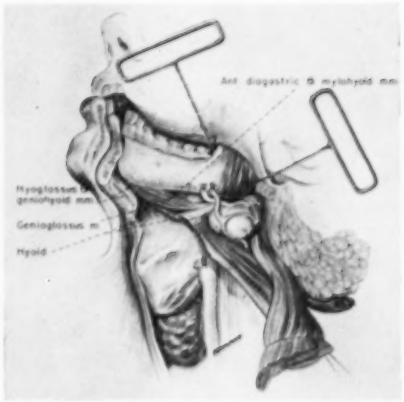

FIG. 13—Combined, in-continuity resection completed; mucosal surfaces and pharyngeal aponeurosis about to be closed; tongue repaired.

placed on each side of the midline near the tip. The distribution of the lesion will govern the extent of resection.

Suturing the severed margin of buccal mucosa is begun posteriorly and continued forward, approximating buccal mucosa successively to the mucosa of the severed root of the tongue and to the floor of the mouth and incorporating the severed margin of the superior limit of the pharyngeal aponeurosis into the closure whenever possible. Repair of the apex of the tongue is accomplished by approximating the superior to the inferior mucosal surfaces (fig. 14).

Thus, in the radical neck dissection alone, by utilizing the principle of splitting the hyoid bone while leaving its muscles attached and carrying out the dissection in the retrohyoid cleavage plane, a single bloc is resected in continuity consisting of all the nonvital structures of one side of the neck from the midline to the free margin of the trapezius and from the clavicle upward to the level of the mandible (figs. 15 and 16). In the commando procedure, the upper limit for dissection is extended to incorporate the hemimandible in this

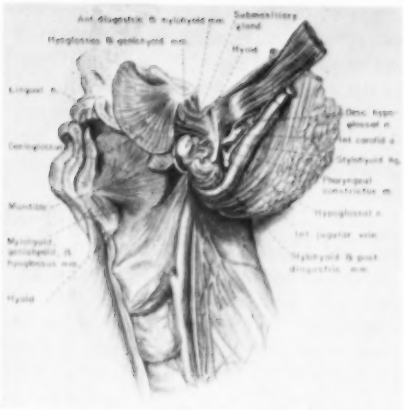

FIG. 14—Internal aspect of specimen after extended retrohyoid commando resection.

FIG. 16—The superficial aspect of the resected specimen after commando procedure.

FIG. 15—External aspect of specimen after retrohyoid commando resection.

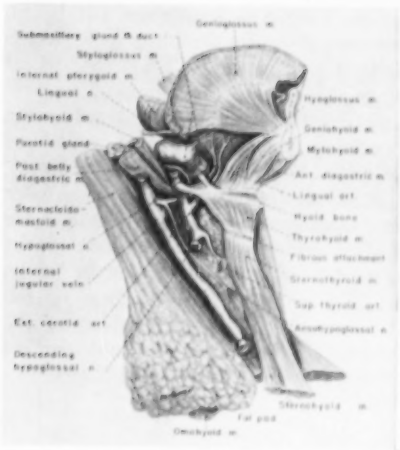

FIG. 17—The deep aspect of the resected specimen after commando procedure.

single bloc as well as the structures of the mouth, as required, and its floor. The dissection in the lower limits extends to the depth of the third layer of the deep cervical fascia, and in the upper limits to the depth of pharyngeal aponeurosis.

The superficial wound is closed in layers, with through-and-through perforated plastic drainage tubes extending from one end of the incision near the temporomandibular fossa to the other, and a separate similar drainage tube brought out at the lower end of the vertical incision. Pressure dressings are then applied.

After an immediate postoperative period of tube feeding, swallowing is normal. The resection of the ribbon and suprahyoid muscles on one side do not affect it, since the muscles of the oppo-

site side are adequate for elevation of the larynx, which accompanies the act of swallowing. I have not included the ribbon muscles of both sides in bilateral radical neck dissection and therefore do not know what the effect upon swallowing might be.

Criticism of so-called needless sacrifice of tissue (particularly of the more superficial muscles of the floor of the mouth when the dissection is limited to the neck) seems unjustified, since it produces no obvious physical disability and such sacrifice for the purpose of obtaining anatomic continuity is entirely in keeping with the principles of good cancer surgery. In the limited procedure when the hypoglossal nerve is sacrificed, the resultant motor paralysis of one side of the tongue is unpleasant but by no means sufficiently disabling to be worth serious consideration when life is at stake. This nerve can, however, be preserved without otherwise limiting the operation. Apart from this, unilateral extended retrohyoid radical neck dissection, as described, produces no greater postoperative disability than the more limited procedure. This applies to the neck dissection alone as well as to the en bloc in-continuity resection of the contents of the neck, the floor of the mouth, the hemimandible and the involved structures of the oral cavity.

SUMMARY

A technique for performing extended radical neck dissection with or without hemimandibulectomy, including resection of involved structures of the oral cavity, is described. This technique is based upon the premise that the hyoid bone is the surgical keystone of the neck and that removal of one lateral half of this structure, allowing the muscles to remain attached, simplifies and permits an en bloc dissection of all structures between the trapezius and the midline from the mandible to the clavicle to the depth of the third layer of the deep cervical fascia. Hemithyroidectomy, permitting the half gland to remain attached to the specimen by the superior thyroid vessels, has recently been added as a routine procedure. The lymphatics concerned in and about the midline of the neck are described.

The extended technique is considered justified (1) because of the lymphatics present in the region of the midline of the neck, which are not ordinarily resected, (2) by examples cited in the literature of frequent recurrences in the immediate area of the operative field as well as in the lymph nodes on the opposite side of the neck, and (3) by virtue of the biology of the tumors, which ordinarily limits them to the area above the clavicle without spread to distant organs, so they can almost always be surgically resected provided the field of neck dissection can be enlarged to include all possible locally involved areas. Extended retrohyoid radical neck dissection is proposed as a technical advance in this direction.

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DISCUSSION

ARNOLD J. KREMEN, M.D., (by invitation), New York: I want to thank you for the privilege of hearing this paper and of making a few remarks on this very interesting subject.

I think Doctor Pressman is to be commended for calling our attention to a very worthwhile extension of the resection of cancer-bearing tissue in the neck.

We speak of radical neck dissection, and it is always, I think, a bad policy to use the term when describing a procedure. What are we going to call Doctor Pressman's operation now, after we have used up our ammunition in describing a lesser procedure as a radical procedure? For extension of radical mastectomy the term superradical mastectomy has come into use. I suppose here, Doctor Pressman, it might be wisest to call this a complete dissection and let it go at that.

This procedure is a valid anatomic extension in the management of cancer problems in this area. I believe that the cardinal reason for the superiority of surgical neck dissection over radiation therapy for management of metastatic cancer to the lymph nodes in the neck is well brought out in this instance, namely a complete extirpation en masse of all lymphatic and potentially cancer-harboring tissue in the area. In the management of lesions of the head and neck, the one concept on which there is wide agreement is that if one is concerned with curing an individual who has metastatic cancer, the surgical extirpation of all the lymph nodes is the only method that will offer a chance of cure. Radiation therapy may be palliative but it is rarely curative, and I think the reason it is rarely curative is because the radiotherapist cannot deliver a cancerocidal dose of roentgen rays to the entire potential area where cancer may reside in the neck; that is, from the clavicle to the mastoid process. He may manage the grossly palpable areas, but this alone does not produce permanent cure. By the same token, I am sure that surgical management of cancer in the lymph nodes would fail if we attempted to remove only the palpable lumps. The reason we have successes is that aggressive surgeons have been able to apply classical concepts of surgical management in this area, namely, complete resection of all potential cancer-bearing tissue in the area.

You have had described to you today an extension of this concept, which removes one more potential area where residual cancer could remain. For this reason I think it wise, and I am certain it will not add to the mortality of the procedure. Certainly it does not appear to add to the technical difficulty of the procedure, and if it does not contribute to added postoperative morbidity, I think many surgeons will add it to their armamentarium.

PRESERVATION OF HEARING IN MASTOID SURGERY: TECHNICAL CONSIDERATIONS

SHIRLEY HAROLD BARON, M.D.
SAN FRANCISCO, CALIFORNIA

CONSERVATION of hearing in surgery for chronic mastoid disease should be a goal, not just for the occasional so-called suitable case but for all cases requiring such surgery. True, the goal will not always be reached, but the incidence of success will be gratifyingly high.[3,4]

Each mastoid operation should be planned as though it were to be done on the patient's only ear with good hearing; then one would have no choice but to be as conservative as possible. With the current refined techniques and modern antimicrobial therapy, this conservatism will result, in most instances, in a useful as well as a dry ear without the hazard of intracranial complications. In my experience an intracranial complication has never occurred as the result of "conservative surgery."

What should the plan be? What must be done to preserve or improve hearing? The most important factor seems to be to maintain a freely movable stapes.[13,14] Next, a patent unscarred round window must be included in a sealed tympanic space connected with a patent eustachian tube. The very best results, with some exceptions, are in those cases in which there is an intact ossicular chain; however, in the absence of the incus and the head of the malleus, serviceable hearing can be obtained if there is a sealed tympanic space.[8,10,13] If this tympanic space should become obliterated by eventual complete retraction of the tympanic membrane against the promontory, however, the good hearing level previously obtained may be lost.[8]

When the incus and the head of the malleus have been removed, the remaining tympanic membrane often will rest against and envelop the capitulum of the stapes. In the cases of mine in which this has occurred and a tympanic air space has been maintained, *a good hearing result has been the rule.*** This has been noted also by Juers, who also feels that this contact between the stapes and the pars tensa is desirable for good hearing. Juers has devised a procedure, the myringostapediopexy,[9] to ensure this contact, which apparently produces a difference between the sound pressures on the oval and round windows which is conducive to good hearing.

We must aim then (1) to maintain the mobility of the stapes within a sealed tympanic space in which there is continuity between the eustachian tube and the round window area; (2) to prevent obliteration of this sealed tympanic space; (3) to ensure contact between the tympanic membrane and the head of the stapes when the incus or the incus and the head of the malleus have been removed.

To maintain stapes mobility, one should take the following precautions:

1. Avoid disturbing an intact functioning ossicular chain when one is present.
2. Avoid dislodging the stapes from the oval window by refraining from

From the Department of Surgery, Division of Otolaryngology, Stanford University Medical School, and the Department of Otolaryngology, Mt. Zion Hospital, San Francisco, California.
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**Footnote, p. 368, and paragraph 2, p. 391, of reference No. 4.

excessive manipulation about it, such as may occur if an attempt is made to remove every bit of granulation tissue surrounding the stapes in its pelvis ovalis (fossula fenestrae vestibulae). It is not necessary to remove every vestige of granulation tissue as the formation of granulation tissue is reversible.

3. Avoid dislodging the stapes by not attempting to remove an enveloping cholesteatoma matrix if one is present.
4. Avoid removal of the mucous membrane in the pelvis ovalis near the fenestra,[14] as this may cause excess scarring and stapes fixation. (Refrain, also, from removal of mucous membrane about the round window to avoid scarring of the round window.)

How can obliteration of the tympanic space be avoided? Some suggestions follow.

1. Preserve the ossicular chain when possible.
2. If the matrix of a cholesteatoma is sealing off a tympanic space, do not disturb the matrix.[2,3,4]
3. If it is necessary to remove the incus and head of the malleus, complete retraction of the tympanic membrane against the promontory with obliteration of the tympanic air space may occur. To prevent this, perhaps one should try to leave behind some "tenting-out" structure, or structures, such as (a) the greater and lesser tympanic spines (see fig. 1), as suggested by Lempert,[11] or (b) the head of the malleus, if it is possible to save this when the incus is removed.

The latter alternative was adopted in case 4, to be presented (fig. 13), and, I believe, definitely contributed toward the prevention of complete retraction of the tympanic membrane in this instance.

FIG. 1—A diagrammatic representation of the greater tympanic spine (1) and the lesser tympanic spine (2). Lempert suggested that leaving these spines may be a way to prevent medial retraction of the tympanic membrane. (Broken lines indicate location for incisions for Lempert-type tympanomeatal flap. See figure 11-A)

If a sealed tympanic air space has been obtained but the expected good hearing has not, one may resort to fenestration of the labyrinth with a reasonably good chance for success, providing that the cochlear function is adequate.

TECHNICAL CONSIDERATIONS

Under consideration are the techniques for all types of surgery for chronic mastoid disease. It is important to use magnification in all instances.

If a modification of the classic complete radical operation is to be done, it does not necessarily have to be the Bondy operation, although the Bondy is the one done most often in this country. There are some cases which need a little more intervention than is usual in the Bondy operation but something less than the completely radical operation. To distinguish these "in between" procedures from the Bondy operation, a term such as "conservative radical mastoidectomy" may be desirable.

The techniques used must fit the situations encountered. A few of these which

have been used successfully to produce a useful as well as a dry ear will be described. In the Bondy operations the "bridge" has been removed in all instances, a detail which, in my opinion, is important. (The technique of the Bondy operation will not be given, as it has been described elsewhere.[2,4])

SITUATIONS ENCOUNTERED

I. *Attic or attico-antral cholesteatoma with attic or posterosuperior-attic tympanic membrane perforation*

a. *Technique when the cholesteatoma matrix is preserved*

Most cases of chronically discharging ears that require surgery have cholesteatomas and a high percentage of these are from attic perforations (fig. 2), or posterosuperior marginal perforations, or a combination of both. Many of these have serviceable, or close to serviceable, hearing. It is expected, therefore, that there is a good chance to maintain or improve the hearing. If the patient's middle ear cavity is sealed off by the matrix of the cholesteatoma, it is rare for the hearing level to drop after surgery, providing that the covering matrix is preserved and the middle ear cavity is undisturbed. Actually, many ears obtain an improvement in hearing, apparently as the result of the elimination of

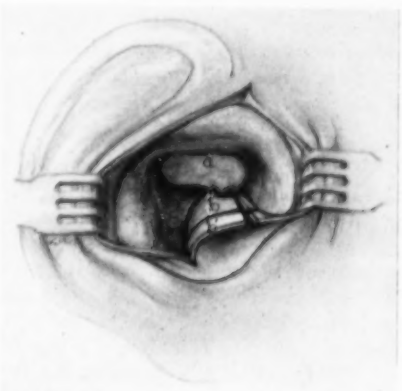


FIG. 3—The outer portion of the cyst has been cut away and the inner layer (known as the matrix) is left undisturbed, *a*. Note that the incision in the long axis of the membranous canal is cut through the perforation, thus eliminating the perforation. The skin flaps *b* and *b'* can now be reflected into the mastoid bowl. (From Baron.[4])

FIG. 2—Diagrammatic representation of a cholesteatoma, which in reality is a cyst, *a*, with a squamous cell epithelial lining on the inside and a connective tissue layer on the outside. It is connected by a stalk to the perforation which, in this case, is in Shrapnell's membrane. (From Baron.[4])

FIG. 4—The completed operation: *a*, the cholesteatoma matrix left in situ. (Note that it is covering the ossicles and is sealing off the tympanum.) *b* and *b'* are the membranous canal skin flaps. (From Baron.[4])

the middle ear congestion after the cholesteatoma is exteriorized. A number of these cases, preoperatively, have a serous otitis media which disappears after surgery, usually spontaneously, although occasionally drainage by myringotomy is necessary.

The matrix cannot be preserved in all cases, as there are often definite contraindications for preserving the matrix. These were outlined in my discussion before this group two years ago.[3]

When the matrix is saved, an advantage gained is elimination of the attic perforation, since the canal skin is not needed for a tympanomeatal flap. This membranous canal wall is cut through its long axis superiorly to and through the perforation, thereby eliminating the perforation (fig. 3). This forms a plastic flap which can be reflected over the facial ridge to the mastoid bowl (fig. 4). For better conformity and maneuverability, an external "cuff" (b′ in fig. 4) should be separated from the main flap.

Should the preserved matrix of the medial attic wall be elevated in order to inspect the ossicles? That depends upon the preoperative hearing level. If the hearing is serviceable, especially if it has decreased only 5 to 15 decibels, it is unwise to disturb the matrix. If the hearing is poor, then the matrix should be elevated and the ossicles inspected.

ILLUSTRATIVE CASE REPORTS

Case 1—This type of procedure was followed for O. G., a 36-year-old male, who had an attic perforation and an atticoantral cholesteatoma in his only good ear, the left. A slight improvement in hearing was obtained postoperatively. A postoperative audiogram, done five years later, showed maintenance of his hearing improvement (fig. 5).

Comment: At surgery, would one dare manipulate the patient's middle ear structures and gamble on the future hearing in his only useful ear? Utilization and preservation of the matrix that

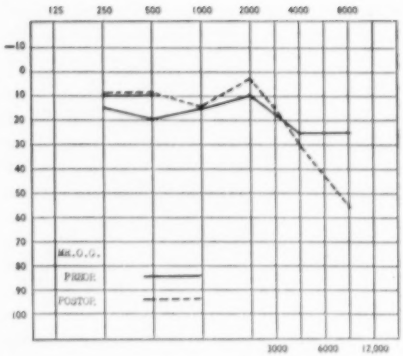


FIG. 5—Preoperative and postoperative audiograms of the left ear, case 1.

sealed off the ossicles and the tympanum was the solution to the problem. One can expect that in this procedure there will be no further hearing loss. In cases previously reported,[3] none that had serviceable hearing before operation lost this hearing when the matrix was saved, and some had further hearing improvement. Also, some patients whose hearing was below the serviceable level showed improvement in hearing to the serviceable level after surgery.

Of the cholesteatomas encountered, the attico-antral one, such as was present in this patient, is found most often.[3] It is obvious, therefore, *that this technique of preserving the matrix is one of the most valuable in the conservation of hearing.*

Case 2—In this case, that of Mrs. L-l, age 34, partial removal of the matrix was required. This left the short process and some of the body of the incus exposed (fig. 6). A flap was made to cover the bare incus and the uncovered portion of the tympanum in the following manner:

A Lempert-type tympanomeatal flap was developed as usual. When this was done, the anterior auditory canal wall attachment was severed in the direction of and through the attic perforation, thus eliminating the perforation (fig. 7). The flap, now attached by a

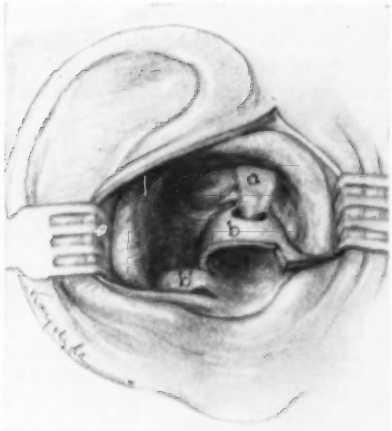


FIG. 6—Technique used in case 2. Part of the cholesteatoma matrix, *a*, covering the head of the malleus, part of the body of the incus, and part of the tympanum were left in situ. It was necessary to remove the posterior portion of the matrix, leaving the short process and part of the body of the incus uncovered. An external cuff, *b'*, cut off from the membranous canal, *b*, has been reflected into the mastoid bowl. (From Baron.[4])

FIG. 8—The completed operation. Note how the skin flap, *b*, covers the incus and the previously uncovered tympanum. It is closely approximated to the edge of the undisturbed matrix, *a*. Primary union occurred between these edges in case 1 and others previously reported. (From Baron.[4])

FIG. 7—The solid and broken lines starting from the floor of the membranous canal indicate the incisions to be made for the flap desired. Note that the anterior incision is carried through the pedicle and through the perforation, thus eliminating the perforation. (The anterior incision through the anterior pedicle is more clearly illustrated in figure 11.) (From Baron.[4])

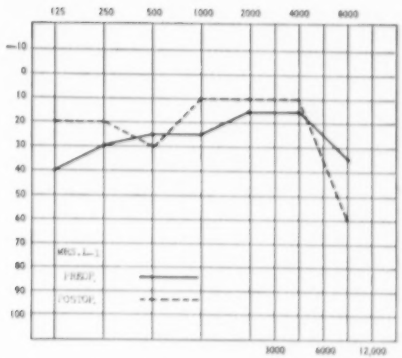


FIG. 9—Preoperative and postoperative audiograms of the right ear, case 2.

single posterior pedicle, was reflected to cover the bare portion of the incus and the uncovered portion of the tympanum, with its anterior free edge closely approximating the free edge of the remaining attic portion of the matrix (fig. 8). It was held in place with paraffined mesh gauze, which was removed in one week. *Primary union* occurred at the margins of the free edges. Hearing improved from a 21.6 to a 16.6 decibel level in the only good ear of this patient (fig. 9). A similar procedure has been done in two other cases, in which primary union and hearing improvement were also obtained.

Comment: It is noteworthy that in this case granulations under the incus and encompassing the stapes were observed but were left undisturbed. A small perforation of the posterior inferior quadrant of the tympanic membrane developed. The middle ear drained through this perforation, but drainage ceased after two weeks and the perforation closed. Ultraconservatism in operating upon the patient's only good ear was rewarded with a hearing improvement.

b. Technique when the cholesteatoma matrix is removed

If one chooses to remove the matrix in cases of attic or attico-antral cholesteatoma with attic or posterosuperior-attic tympanic membrane perforation, it will be necessary to cover the epitympanum with skin in some manner. If one uses a Lempert-type tympanomeatal flap, the flap will contain a perforation which, in about 50 per cent of the cases, will close spontaneously. Those cases in which the perforation does not close will develop an attic fistula, with a 25 per cent incidence of constant or intermittent drainage.[3] An unhealed attic fistula is a potential source of a new cholesteatoma; therefore, if one uses a tympanomeatal flap with a perforation, one should try to eliminate the perforation by one of several methods:

1. Split the flap from the perforation to the superior edge of the flap and freshen the margins of the perforation; then approximate the edges by gentle teasing together and hold them in place with paraffined mesh gauze (fig. 10). In addition to closing the perforation, this will seal the tympanum.

2. Another method calls for the development of a Lempert-type tympanomeatal flap, severing the anterior attachment in such a manner that the incision reaches the perforation, thus eliminating the perforation (fig. 11-A). This leaves a very mobile flap on a very small posterior pedicle, which can now be rotated to cover the tympanum. It is rotated enough anteriorly to completely seal the tympanum. The margins of the perforation should be freshened before approximating the flap.

Such a flap was used in case 3*, that of a 50-year-old female. In this case it was felt necessary to remove the matrix because it was entwined between and around the incus and the head of the malleus. The incus and the head of the malleus were also removed. Complete closure of the tympanum was obtained, but there was a *loss of hearing* from a 6.6 decibel to a 33.3 decibel level (fig. 12).

Comment: While this case illustrates how the tympanum can be sealed with a non-perforation-containing flap, it demonstrates also that one cannot always be sure of retaining hearing when the incus and the head of the malleus are removed. There has been a growing tendency to sacrifice the incus and the head of the malleus. This should not be done when it can be avoided since, as this case illustrates, there is the possibility of a hearing loss.

3. If one chooses to reflect the membranous canal as a flap into the mastoid bowl instead of developing it into a tympanomeatal flap, the tympanum can be sealed by a thin skin graft, either in the presence or in the absence of the incus and the head of the malleus, as suggested by Meltzer,[15] who stated that this graft should extend one millimeter over the upper edge of the tympanic membrane.

4. There is another method of sealing the tympanum, described by Martin earlier this year, to which you are referred.[4]

*Cases 2 and 3 are from the Letterman Army Hospital. They are presented through the courtesy of Col. Byron G. McKibben, Chief of the Ear, Nose and Throat Service, and the courtesy of the Surgeon General of the Army.

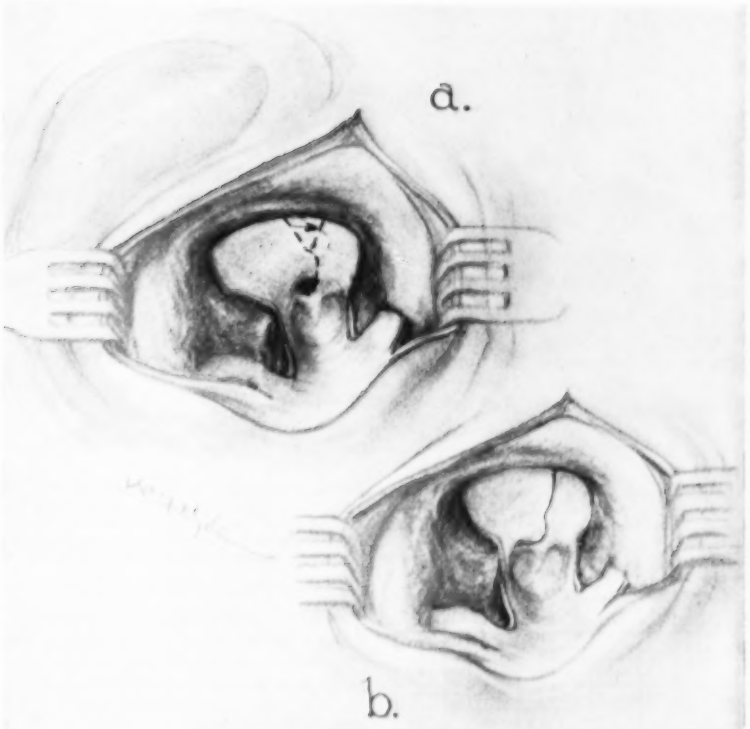
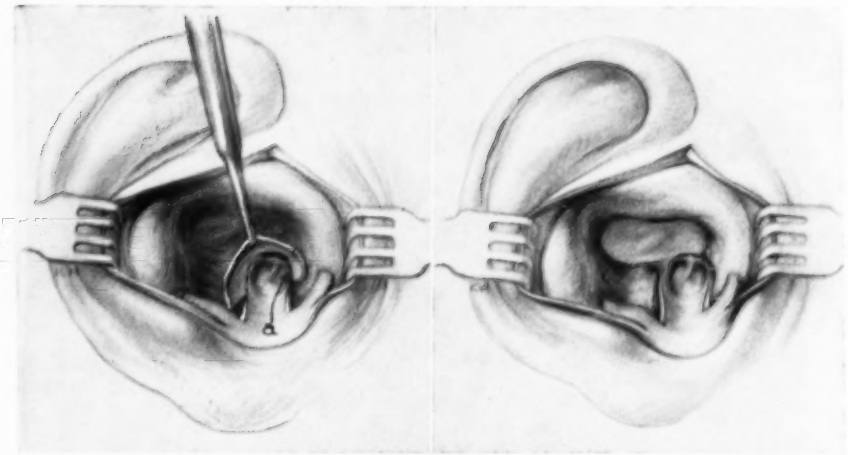


FIG. 10—*a*. If a typical Lempert-type tympanomeatal flap is used in the presence of an attic perforation, there will remain a perforation within this flap. The broken lines suggest a method of incision to rotate the posterior half of the flap forward and downward to eliminate the perforation. (The margins of the perforation are freshened before this is done.) *b*. The perforation is closed. (From Baron.[4])

II. *Large marginal or nonmarginal posterosuperior tympanic membrane perforation without cholesteatoma*

In the past, some cases in this category were treated surgically by an extensive cortical (simple) mastoidectomy. With the current techniques, better results, both in hearing and in freedom from discharge, can be obtained by the modified radical operation, due to better exposure, more complete eradication of the disease and elimination of "dead" space; and by the complete removal of the external attic wall and "bridge," a better "draping" of the skin in continuity with the tympanic membrane onto the medial attic wall will occur. This increases the chances for closure of the perforation and the development of a sealed tympanic space with resultant better hearing.

The possible management of such a situation is best described by reporting case 4, that of a 14-year-old girl who had a large posterosuperior marginal perforation in her left ear with a chronic foul-smelling discharge and an average hearing loss of 50 decibels. A Bondy type of endaural, modified radical mastoidectomy with a Lempert tympanomeatal flap was done. A considerable amount of bony erosion was noted in the mastoid antrum and in the solid angle. No cholesteatoma was found.



11-A 11-B

FIG. 11—A. A method of elimination of the perforation in a Lempert-type tympanomeatal flap. The anterior pedicle is severed at *a*, to and through the perforation. (See figure 1, broken lines, for initial incisions of a Lempert-type flap. See figure 11-B for flap in its final position.) B. The flap in position attached by a thin posterior pedicle. (The upper edge of the tympanic membrane is "freshened" before the flap is placed.) This type of flap may be used to cover and seal the tympanum either in the presence or in the absence of the ossicles.

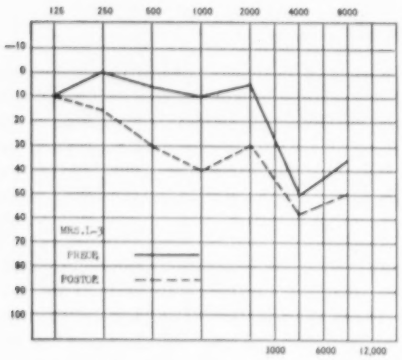


FIG. 12—Preoperative and postoperative audiograms of the right ear, case 3.

The entire mastoid and the middle ear, including the pelvis ovalis and the stapes, contained abundant granulation tissue. The incus was deformed, with some loss of both long and short processes. The incus was removed, but the head of the malleus was left in situ (experimental, in an attempt to prevent medial retraction of the tympanic membrane). All granulation tissue in the mastoid and epitympanum, and as much as possible in the pelvis ovalis, was removed, but that surrounding the stapes was left undisturbed. It was felt that the granulation tissue might disappear and that the tympanic membrane perforation, although large, might close under treatment. The granulation tissue did disappear and the perforation, with treatment (cauterizing the edges with trichloracetic acid and patching with Cargile membrane), eventually closed. The new membrane closely hugged the capitulum of the stapes and the promontory, but the anterior half of the tympanic membrane *did not* retract to the medial wall (fig. 13). The hearing improved to the 18 decibel level, an overall improvement of 32 decibels (fig. 14).

Comment: This case proves the following important points:

1. Granulation tissue formation is reversible.
2. A large, marginal tympanic membrane perforation can be closed in an operated ear. It has not been possible, thus far, to close a marginal perforation in a nonoperated ear.[1]
3. The head of the malleus can act as a protuberance to "tent out" the tympanic membrane sufficiently to pre-

FIG. 13—Appearance of the healed tympanic membrane in case 4. The capitulum of the stapes is closely enveloped by the new membrane of the healed perforation. The retraction of the new membrane against the medial wall is evident. The anterior one-half of the tympanic membrane has not retracted (retraction probably prevented by preservation of the head of malleus). The contact of the tympanic membrane against the mobile stapes in a sealed tympanic air space has resulted in excellent hearing.

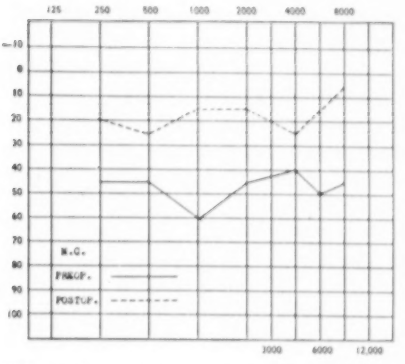


FIG. 14—Preoperative and postoperative audiograms of the left ear, case 4.

vent its complete retraction against the promontory.

4. The pressure of the tympanic membrane against the capitulum of the stapes is conducive to a good hearing result, provided that there remains an air space in the sealed tympanum.

III. *When the tympanic membrane is absent and the middle ear is occupied by a cholesteatoma*

In cases falling in this category, the surgical technique differs from the classical radical mastoidectomy only in that the matrix, which is firmly approximating the stapes, the promontory and the eustachian tube orifice, is not disturbed. This technique is especially desirable in those cases which have a fair amount of residual hearing, such as was present in case 5.

Case 5, a 14-year-old boy, on whom this type of conservative radical mastoidectomy was done. An audiogram one and one-half years postoperatively showed a very definite hearing improvement. What was left as cholesteatoma matrix now has the appearance of a thin pseudo drum membrane closely hugging the head of the stapes and enclosing a tympanic air space. The chances for a hearing improvement would have been considerably less had the cholesteatoma matrix been removed (fig. 15).

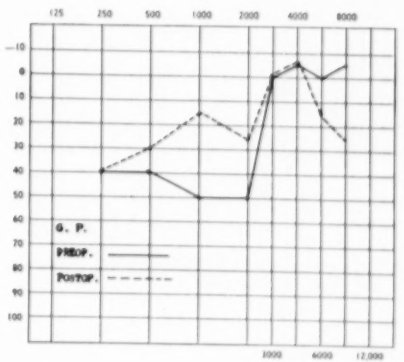


FIG. 15—Preoperative and postoperative audiograms of the left ear, case 5. After healing, the matrix was seen to be changed from a "stiff," thick, white scaly structure to a clean, pink, thin, pliable skin. At first, no tympanic air space could be discerned, but after one year and eight months, this new pseudo drum membrane had "lifted" from the lower portion of the medial wall and the hypotympanic air space became visible.

IV. *When the tympanic membrane is absent, destruction is severe and the classical complete radical mastoidectomy is necessary*

The purpose of presenting this classification is to report unexpected good hearing results in two cases in which the complete radical operation had been done. Each developed an unplanned

pseudo drum membrane with a sealed tympanic air space in continuity with the eustachian tube.

In one (case 6), where the pseudo drum membrane can be seen closely enveloping the head of the stapes, the hearing improved from a 38 decibel to a 3.3 decibel level (fig. 16). In the other (case 7), the hearing level improved from 60 to 21.6 decibels (fig. 17). In the latter, the head of the stapes cannot be seen, but it is most probable that it is in contact with the pseudo drum membrane.

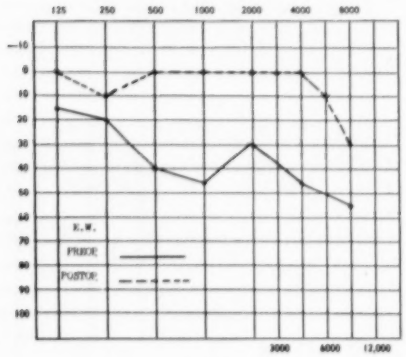


FIG. 16—Preoperative and postoperative audiograms of the right ear, case 6.

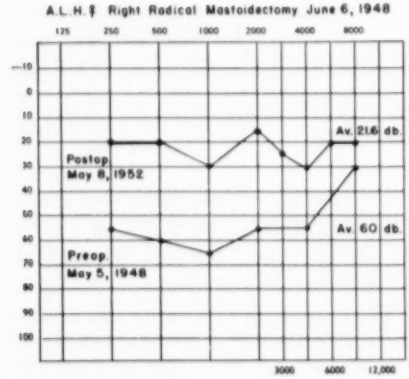


FIG. 17—Preoperative and postoperative audiograms of the right ear, case 7.

In a third case (case 8) which healed with a pseudo drum membrane and a tympanic air space but not with a good hearing result, I did, experimentally and with the patient's knowledge that it was experimental, a fenestration operation. I had not performed the previous radical operation, so I had no knowledge of what middle ear manipulation had been done. At surgery a tympanic air space was found, but there was considerable scarring in and about the round window. Nevertheless, I fenestrated the horizontal semicircular canal. Good hearing was not obtained, but there was some hearing improvement (fig. 18) which the patient feels is of distinct benefit.

The presentation of this case is not intended to create the impression that fenestration of the labyrinth following radical mastoid surgery can be done with abandon. It is reported to illustrate further the many avenues to function that are opened when healing with a sealed tympanic air space is obtained.

Comment: The results in the last three cases suggest that perhaps we should change the perspective in our techniques and aims, even with the complete radical operation. I can recall being taught that in the healing of a radical operation, the formation of a pseudo drum membrane with an air space in back of it should be avoided; now I

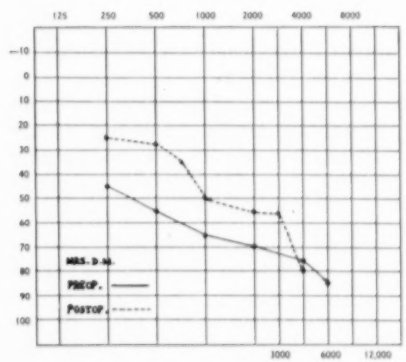


FIG. 18—Preoperative and postoperative audiograms of the right ear, case 8.

should like to know how to produce it regularly.

Certainly, as pointed out by Meltzer,[14,15] traumatizing the oval and round window areas should be avoided, and, as Day[5] stated, drum membrane remnants should be utilized rather than destroyed. Perhaps the bony annulus should be preserved and a skin graft applied after the method of Martin,[4] House,[6,7] or Sullivan[16]; and maybe one shouldn't be so anxious to seal off the eustachian tube if a tympanic air space is a requirement for hearing.

DISCUSSION

It has not been the intent of this presentation to renew the controversy, presented before this organization two years ago, about whether it is safe to preserve the matrix of a cholesteatoma.[3,12,17] There are certain situations, previously described, in which it is definitely safe to preserve the matrix, and in these cases the over-all best hearing results will be obtained.

In recommending that granulation tissue about the stapes be undisturbed because it is reversible, I have deviated somewhat from the usual teaching. In a few cases, when I was trying meticulously to remove the granulations around the stapes, I either dislodged or tilted the stapes to the detriment of the hearing, so I felt that perhaps I should be just a little less bold when working in this area. I have left the granulation tissue about the stapes in enough cases to know that when the main mass of granulation tissue in the mastoid and epitympanum, with its adjacent infected bone, is removed, the small amount of granulation tissue which is left behind disappears. This conservatism has been rewarded with very good hearing and dry ears, as evidenced by case 4, herein reported, and by others in my files.

It must be emphasized that the incidence of success, as to both hearing and dryness, will be low following the use of any of these techniques if postoperative care is not meticulous and precise. The control of healing is as important as the operation.

Considerable progress has been made in hearing conservation by improved techniques in the modified or conservative radical mastoidectomy; but, understandably, this has not been so with regard to the complete radical operation. The last three operations reported, all complete radicals (cases 6, 7 and 8), indicate that the formation of a pseudo drum membrane which contacts the head of the stapes and which produces a sealed tympanic air space with a patent round window in continuity with the eustachian tube is desirable for good hearing. How this pseudo drum membrane, as seen in these cases, can be planned for in the routine complete radical operation, is not yet entirely apparent; perhaps in the not too distant future the solution will be at hand, a welcome addition to the many advances that have been made toward salvaging the function of an ear.

SUMMARY AND CONCLUSIONS

1. In surgery of the mastoid for a chronic inflammatory process, it is possible to obtain a good hearing result in all types of cases; therefore, one should aim for a technique that will conserve or improve hearing.

2. This technique should be designed to maintain a mobile stapes and an unscarred round window in a sealed tympanic air space in continuity with the eustachian tube.

3. Techniques that have been successful in accomplishing this have been described. These include the management of various situations, with and without cholesteatoma, with and without preserving the cholesteatoma matrix.

4. In situations where it has been necessary to remove the incus or the incus and the head of the malleus, contact

between the tympanic membrane and the head of the stapes is desirable for good hearing.

5. If a sealed tympanic space has been obtained but good hearing has not, fenestration of the labyrinth may be done with a good chance for success.

6. If, in the complete radical operation, a pseudo drum membrane develops, contacts the head of the stapes and produces a sealed tympanic air space with a patent round window in continuity with the eustachian tube, good hearing will result. Cases have been reported in which this occurred. How to plan for such a pseudo drum membrane is on the agenda for tomorrow.

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DISCUSSION

ARTHUR L. JUERS, M.D., Louisville, Kentucky: Doctor Baron has presented an interesting and important phase of otology in a most adequate manner. It is particularly desirable that the technical aspects of preserving hearing in surgery for chronic attic and middle ear disease be emphasized. There are still too many residents completing their training in otolaryngology without having acquired a comprehensive understanding of this subject.

In general, I find myself in agreement with most of the points Doctor Baron has stressed. He cited one case in which it was necessary to remove a well functioning ossicular chain with a resulting hearing loss of about 25 decibels. I have had a similar experience in a few instances and have suggested a surgical technique which gives virtual assurance of serviceable hearing after removal of an incus.

A few years ago I reported a few instances in which a 20-decibel hearing level existed in

cases in which the incus was destroyed by cholesteatoma, which had simultaneously displaced the posterosuperior portion of the pars tensa medially into contact with the stapes. Doctor Baron has substantiated this observation. It occurred to me that when the removal of attic disease necessitated sacrificing a functioning incus, a good hearing level after surgery could be assured if a similar relationship were established at the time of the operation. I have termed this procedure "myringostapediopexy," indicating contact between the tympanic membrane and stapes.

If there is smooth matrix overlying the ossicles, sealing off the tympanic cavity, I believe it is advantageous not to disturb this area. However, in these cases I do remove the matrix superior and posterior to the prominence of the lateral semicircular canal, because I have in several cases found in this area unsuspected finger-like projections of the matrix toward the petrous pyramid. If cholesteatoma surrounds portions of the ossicles, or incompletely seals off the tympanic cavity, I usually remove all matrix. I have found a well functioning ossicular chain in only 15 to 20 per cent of these cases. (Slides demonstrated the steps in the technique of myringostapediopexy.) If the surgeon prefers, the tympanic cavity could be sealed off with a skin graft. However, a failure of the graft to take would result in an attic fistula. Dr. Baron has already mentioned that a residual attic fistula is conducive to further otorrhea and poor hearing.

I would like to comment on the statement which Dr. Baron made regarding the desirability of retaining the head of the malleus when the incus is removed. He feels that retaining the malleus head lessens the likelihood of retraction of the pars tensa and its adherence to the promotory after surgery. I have had only one instance in which the tympanic cavity was obliterated after surgery. In this case I removed the entire malleus because of a pocket behind the manubrium. I have always removed the malleus head when the incus is absent and have noted no tendency for the pars tensa to retract as a result. However, the only objection I can make to retaining the malleus head is that it tends to form an elevation in the depths of the cavity. Any irregularity of contour will encourage the accumulation of debris, and where there is debris, there will be a tendency toward surface infection.

Doctor Baron's observations concerning serviceable hearing levels following radical mastoidectomies in which pseudomembranes developed are interesting and contributive. I wonder whether preserving the inferior and anterior portions of the tympanic ring would encourage the development of such a membrane?

Again, I want to commend Doctor Baron on his excellent presentation.

ROBERT C. MCNAUGHT, M.D., San Francisco, California: I was pleased to be asked to discuss Doctor Baron's paper because otology has now come of age and our sole objectives need no longer be the saving of life and the avoidance of intracranial complications. We can now consider the preservation and, in some cases, the actual improvement of function.

The ruthless destruction of functional structures in the name of safety for the patient is becoming the exception, at least out our way, rather than the rule that, of necessity, it once was. We are doing more modified radical operations than we are complete radical operations. Even in those cases where disease has destroyed most of the functional structures, what remains is conserved and utilized. Cholesteatoma, which is nature's attempt at healing, is controlled and is aided in this attempt.

I disagree with Doctor Juers in that I believe that even though the cholesteatoma may extend medial to the ossicular chain, it is not so dangerous when it has been externalized. There have been amazing developments in some of these cases in which the incus was not sacrificed. I have watched some of these patients for long periods. In fact, I have seen cases in which I didn't know how the mechanical connection between the malleus and the stapes came about. There had been an incus at one time. It gradually disappeared but the hearing level remained up, and I am sure that nature in those cases did a better job than I would have been able to do. Even granulation tissue may reverse itself, and it usually does if the surgeon exerts the necessary restraint. It does require restraint, patience, and skill in the postoperative care.

Even in the distant past there were some cases of radical mastoidectomies that fortuitously resulted in amazing hearing levels.

Some of the cases that Doctor Baron reported have been tested at Stanford, and I can verify the audiograms that you saw, the "impossible" hearing level as predicated by our present conception of the mechanics of hearing. A great many people are thinking along the same line, trying to discover just what mechanical setup is present.

I am not at all sure that the beautiful techniques which Doctor Kobrak has shown are actually helpful in this regard, because we are seeing the protective function of the ossicular

chain as well as its function in sound conduction. Nevertheless, we have to understand the normal mechanics in order to control the factors that may produce a good hearing level, and I am of the opinion that there are several mechanical linkages that make possible a good hearing level.

In the modified radical operation we aim to restore the normal ossicular chain mechanism with a sealed tympanic space, but I am sure that this method is not the only one that will preserve a high degree of hearing, because we have seen patients without any ossicular chain who have a high hearing level.

I don't have time to go into some of the theories that I have been pondering, but I do believe that while the impedance matching function of the ossicular chain is important, actually the drumhead and the ossicular chain evolved as a defense mechanism for an extremely delicate organ that has to be subjected to a tremendous range of sound energy. Such a sensitive organ hardly needs a gross mechanical linkage to increase its efficiency. In other words, I don't believe that teleological explanations are without danger, and we have accepted, almost without question, the fact that this system evolved to increase the efficiency of the inner ear. Actually, we know that as soon as the perilymph space is opened under local anesthesia, the patient hears better than he will at any subsequent time. [Doctor Shambaugh subsequently informed me that while similar statements are found in the literature, unpublished experiments of his have failed to support this concept.] A sound-gathering system one hundred times the area of the fenestra is used to concentrate the sound and serve as an impedance matching device; yet, during the fenestration operation it does not serve as well as an opening into the perilymph space.

All this and other conflicting evidence leads me to believe that we may eventually evolve a substitute mechanical system whereby we can obtain excellent hearing levels. Antibiotics and improved techniques now make it possible to anticipate such developments.

DOCTOR BARON: I wish to thank Doctor Juers and Doctor McNaught for their very excellent discussion.

I think Doctor Juers' contribution of the myringostapediopexy is definitely going to be very helpful.

I was glad to have Doctor McNaught as one of the discussers because he has seen my cases and I have seen his, and we have talked this problem over many times.

I want to say, especially to the younger men, that my axiom to our residents-in-training is, "You can always take out more, but seldom can put anything back. It is not a blot on one's escutcheon to have to reoperate a case if the initial surgery, done with the aim to preserve hearing, proves inadequate. This inadequacy, however, must be a planned one and must not be due to inadequate knowledge or inadequate postoperative care. Postoperative care is every bit as important as the operation. If this care is not given, the result may be poor."

SIMPLIFIED TANGENT SCREEN EXAMINATION USING A RECORDING SCOTOMETER

WILLIAM U. MCREYNOLDS, M.D.
ANN ARBOR, MICHIGAN
BY INVITATION

FIGURE 1.

THE measurement of visual fields is often a tedious task for the busy ophthalmologist. This is especially true of detailed tangent screen examination, which may become very time consuming when as many as twenty or thirty points must be indicated and plotted. To facilitate examination with the tangent screen and to plot the results directly, Marks[1] described, in 1921, the use of the pantograph. This is an instrument used by draftsmen for accurately reducing or enlarging drawings. The method evidently proved too cumbersome and distracting to be of value. Posner[2] used the same device, but only to transfer the position of the marking pins to the recording paper.

The scotometer I am presenting was designed to speed tangent screen examination and to plot the results at the same time without distraction to the patient. Essentially, it is a pantograph which operates by fine cables instead of the usual levers (fig. 1). The operator holds the recording pencil, which is on the end of a levered carrier, over the recording paper. Two cables run from the pencil to the axles of two enclosed wheels above the paper. Cables run from these wheels through pulleys at each upper corner of the screen to the test object. The diameter of the axles is to the diameter of the wheels as the width of the recording paper is to the width of the tangent

Instructor, Department of Ophthalmology, University of Michigan Medical School, Ann Arbor, Mich.
Presented as a *Note Instrument* at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

screen. Thus, as the operator moves the pencil over the recording paper, the object moves correspondingly and accurately over the tangent screen. To record the position of the object, the operator needs merely to depress the pencil to the recording paper.

Since the reference points are plotted as the examination is conducted, there is no need for marking pins. The objects are held by a tiny clasp and are readily changed. The fine cables transporting the clasp are practically invisible at one meter so that the usual distraction to the patient of a large object holder and the examiner's hand and arm is eliminated. This scotometer offers not only a simple and rapid means of tangent screen examination but also automatic and accurate charting of the results.

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LIGHT CLIP-ON PLASTIC PRISMS
For the Temporary Correction of Heterophoria and Heterotropia

CONRAD BERENS, M.D.
NEW YORK, NEW YORK

IT has been evident for several years that lighter clip-on prisms which can be changed frequently without too great expense to the patient are needed, especially for patients who have strabismus. Therefore the manufacturer* was asked to make these prisms available for patients who had strabismus.

Having used them with satisfaction, it is hoped that others may also find them useful.

Description

The prisms, which are made of plastic, are 40 mm. in diameter, and each lens is marked with the number prism diopters and the axis. There are six pairs of prisms, two each of 1, 2, 3, 4, 5 and 6△. Stronger prisms are available from the manufacturer.

These are placed in a compact plush-lined box with four clip-on frames and a small screw driver (fig. 1).

Indications for the Use of the Prisms

Both horizontal and vertical heterophoria and heterotropia, up to and including 12△ or more, may be corrected. Combined errors may be corrected by rotating the prisms away from the vertical and horizontal meridians after consulting a prism chart.

How Prisms Are Prescribed

The desired prismatic correction is determined by testing with the Maddox rod, by the screen test with prisms at 6 mm. and 25 cm. and in the cardinal directions of gaze, and by wearing the prisms in a trial frame while the eyes are performing the work for which the correction is to be prescribed.

Aided by the Ophthalmological Foundation, Inc., and the Department of Research, New York Association for the Blind.
Presented as a *New Instrument* at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.
*R. O. Gulden, Philadelphia 20, Pa.

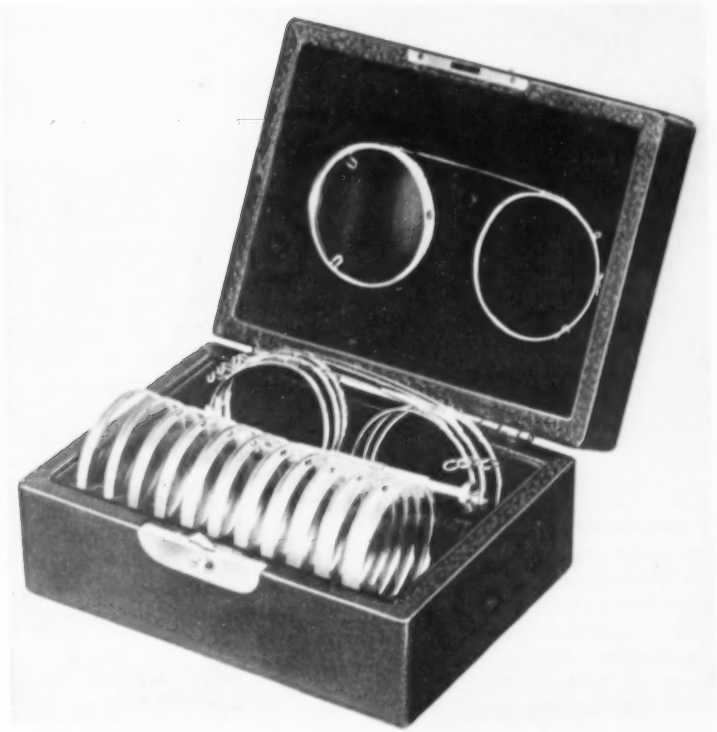

FIG. 1—Six pairs of plastic prisms and four frames in a plush-lined box.

Insertion of the Prism

The screws in the frame are loosened and the prisms are placed in the clip-on frame, which is then applied either to the outside or inside of the patient's spectacles. The four prongs are adjusted so that the prisms are satisfactorily centered.

The prisms may be used many times, as they are indestructible and can be repolished by the manufacturer.

STERIDROPPA

RICHARD C. TROUTMAN, M.D.
NEW YORK, NEW YORK

THE Manhattan Eye and Ear Hospital has used ophthalmic solutions in individual single-dose disposable containers for more than seven years.

At the Annual Meeting of the American Academy of Ophthalmology and Otolaryngology in 1952, we presented a new type of dispenser which would expel an individual dose of sterile ophthalmic solution.[3] While this device had many distinct advantages over any method previously used, there were still some defects which needed further study.

These defects were primarily of a mechanical nature. We found that the original device in some instances allowed evaporation after a period of time in stock, and it was necessary to make changes in the rubber fitments which seal the solutions inside the glass tube. The device I am now presenting overcomes this drawback by a unique plug and valve incorporated as a single unit and providing positive seal against evaporation.

As in previous units, the Steridroppa contains all solutions used in our hospital for preoperative, operative and postoperative medications. The contents are clearly marked in a visible and chemically insoluble ceramic ink, with a distinctive color for each drug. Furthermore, the quality of the rubber used has been improved, and the inside surfaces are now coated with a silicone lining, which prevents contact of the solution with the rubber (fig. 1).

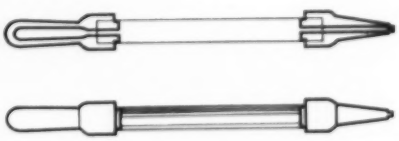

FIGURE 1.

The solutions are especially buffered to preserve the potency of the active drugs, and they have been sterilized according to the standard method suggested by Theodore and Feinstein.[1,2]

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Presented as a *New Instrument* at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

SUPERIOR RECTUS TUNNEL IMPLANT FOR IMPROVING THE COSMETIC RESULT OF ENUCLEATION

RICHARD C. TROUTMAN, M.D.
NEW YORK, NEW YORK

I SHOULD like to present as a new instrument, an implant designed on the basis of experience with the magnetic implant presented before this assembly five years ago. The new implant has been used in too few cases as yet to permit a definitive report. The implant from the anterior aspect is roughly oval. In the upper portion there is a hole which is drilled to accommodate the superior rectus muscle. The face of the implant below this hole is round; it is covered with a stainless steel meshwork, which extends over the edges of the implant for about 3.5 mm. In this area there is a groove to permit the passage of needles. There is an additional groove across the face of the implant to permit the passage of needles in suturing Tenon's capsule. The side view shows the superior rectus tunnel to be roughly in the same curve as the inferior aspect of the implant. The advantages of this modification are (1) the prevention of retraction of the upper lid, producing sulcus as a late complication of absorption of orbital fat; (2) the prevention of rotation should one or more muscles become detached, since the implant is more firmly affixed in the socket, and (3) prevention of retraction and thinning of Tenon's capsule as a late complication. A description of the technique will be published shortly so that those of you interested in trying it may do so. The implant will be supplied with or without magnet, as desired, as perfectly satisfactory motion can be obtained without use of a magnet and many areas do not have facilities for properly placing these in the prosthesis.

Presented as a *New Instrument* at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

AN ADENOID-BITING FORCEPS

FORDYCE JOHNSON, M.D.
PASADENA, CALIFORNIA

A CLOSE examination of the adenoid during every tonsillectomy and adenoidectomy has impressed upon me the need for an instrument to remove remnants of adenoid tissue in this area more adequately. The presence of pieces of adenoid tissue in the higher area of the nasopharynx, and even into the posterior choanae, following an adenoidectomy has been nearly a routine finding, confirmed by the constant use of a large laryngeal mirror to recheck the adenoid area during surgery. The instruments available did not give me ease of access to this area. The adenoid-biting forceps* has been devised for this purpose (fig. 1). It presents a right-angled biting surface, allowing an additional half inch (1 cm.) or more of bite into the region of the choanae and fossae of Rosenmüller. Use of this instrument has resulted in a cleaner adenoid bed at the termination of the adenoidectomy.

FIGURE 1.

The adenoid-biting forceps is also a useful instrument for removing adhesions in the nasopharynx, those on the posterior wall, and those between the tip of the eustachian tube and the vault of the nasopharynx. The preferable technique is to excise such adhesions rather than merely to section them.

The use of the adenoid-biting forceps does not mean exclusion of the use of the adenotome or adenoid curet. Cutting the tissue in this area should be kept at a minimum in order to minimize bleeding. This new forceps is to be used to remove those loose ends of adenoid tissue left after the routine adenoidectomy.

In performing tonsillectomy and adenoidectomy, I sit at the head of the patient and use the Davis mouth gag (with the gag supported by a Mayo stand) and the head-low position, thus draining all the blood toward the nasopharynx. By using suction and a large laryngeal mirror, dipped in green soap and then wiped to prevent fogging, I have a clear view of the nasopharynx. The soft palate is easily elevated by the shank of the adenoid-biting forceps, so that tags of adenoid tissue may be readily picked up.

Presented as a *New Instrument* at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

*The instrument is available through George P. Pilling & Son Co., 3451 Walnut St., Philadelphia, Pa.

MODIFIED HANDPIECE FOR TEMPORAL BONE SURGERY

BEVERLY W. ARMSTRONG, M.D.
CHARLOTTE, NORTH CAROLINA

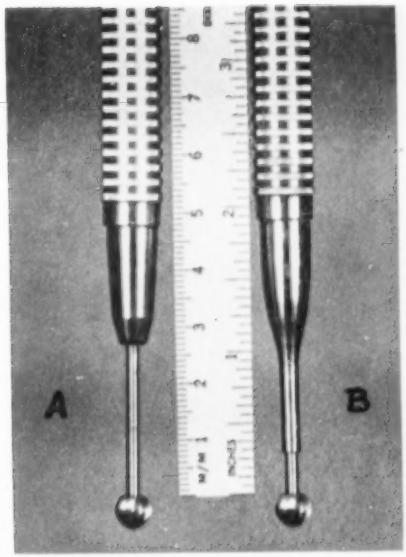


FIG. 1—A, conventional handpiece; B, modified handpiece.

A HANDPIECE used in surgical procedures on the temporal bone has been modified by the addition of a thin metal sleeve that projects from the nose of the instrument. Trauma to soft tissues that results from contact with the rapidly rotating shaft is eliminated by covering the shaft of the surgical bur with the sleeve. The sleeve does not interfere with normal use of the handpiece and does not reduce visibility.

Conventional handpieces leave exposed approximately 27 millimeters of the shaft on the surgical bur (fig. 1, A). The rotating shaft frequently injures skin, soft tissue and cartilage. This trauma is usually minor but it does increase the postoperative discomfort of the patient. In the fenestration operation, there is the added danger that the flap may be avulsed if contact is made with the rotating shaft.

Adding a sleeve* (fig. 1, B) reduces the exposed shaft to approximately 7 millimeters and eliminates completely the trauma to the pinna. The danger of avulsing the flap in the fenestration operation is also reduced to a minimum.

From the Charlotte Eye, Ear and Throat Hospital. Presented as a *New Instrument* at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

*The sleeve must be individually fitted. This one was prepared by the S. S. White Dental Manufacturing Company, Philadelphia, Pa.

A LARYNGEAL DILATING KNIFE

JOHN S. KNIGHT, M.D.
KANSAS CITY, MISSOURI

IN the past, difficulty has been encountered in separating or cutting intralaryngeal adhesions or webs. Various types of long-handled knives have been used. Complete control of the knife blade, especially in maintaining an exact anterior-posterior direction, was not easy for the author. A Jackson triangular laryngeal dilator with a small sharp knife blade incorporated in the superior or apical border has solved our problem of incision and dilatation (fig. 1). The instrument is made by George P. Pilling & Son Company.

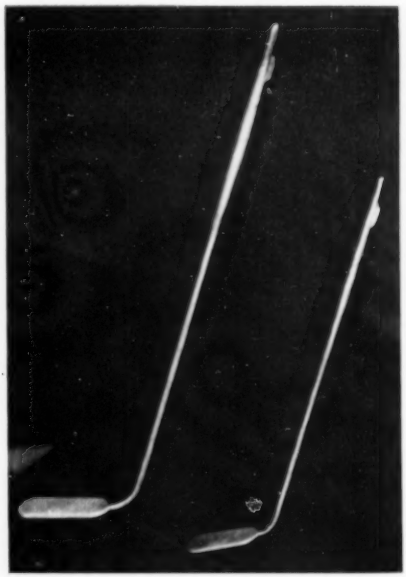


FIGURE 1.

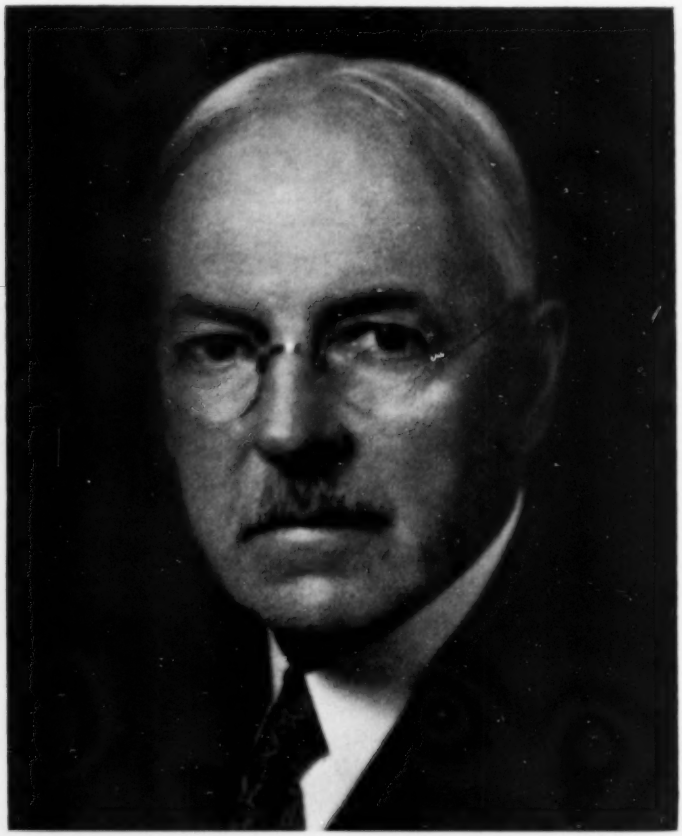
Presented as a *New Instrument* at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

CHANGE OF ADDRESS

PLEASE NOTIFY the office of the Secretary of the Academy of change of address. TRANSACTIONS mailing envelopes are addressed three weeks before mailing date. Your copy will reach you if we have your correct address. Please keep us up to date on your CITY, ZONE AND ROOM NUMBER OF YOUR OFFICE BUILDING. TRANSACTIONS and DIRECTORIES are returned to the publisher each month because we have not been informed of changes of address.

The DIRECTORY is compiled once a year, in March. Check your name and address now to see if it is correct. If not, please notify the editor at once. Send us word immediately of any future change.

In Memoriam



WALTER ROBERT PARKER, M.D., Sc.D.
1865-1955

Dr. Walter Robert Parker died April 1, 1955, at his home in Grosse Point, Michigan, after an illness of many years standing. Doctor Parker was born October 10, 1865, in Marine City, Michigan, the son of Leonard Brooks Parker and Jane Sparrow Parker. He married Margaret S. Watson on December 28, 1907, and there were no children. Mrs. Parker died in 1936.

Doctor Parker's preliminary education was received in the Marine City Public Schools. He attended the Michigan Military Academy in 1883. In 1888 he received his Bachelor of Science degree from the University of Michigan, and his degree in medicine was conferred by the University of Pennsylvania in 1891.

Doctor Parker's postgraduate training was extensive for his time. He was intern at St. Joseph's Hospital, Philadelphia, in 1891-92. He then interned in Children's Hospital, Philadelphia, the following year (1892-93). Ophthalmological training was obtained by a year (1893-94) at Wills Eye Hospital and postgraduate study in Vienna in 1896.

Doctor Parker then moved to Detroit, Michigan, where he opened an office, limiting his practice to diseases of the eye and ear. He became Clinical Professor of Ophthalmology in Detroit College of Medicine in 1899 and remained at this post until he was appointed Clinical Professor of Ophthalmology in the University of Michigan in 1905. He was appointed Professor of Ophthalmology, University of Michigan, in 1907 and held this post continuously until 1932, when he retired from the University as Emeritus Professor of Ophthalmology. During his association with the University of Michigan, he conducted a part-time practice in ophthalmology in Ann Arbor and Detroit. He was consultant in Ophthalmology at Harper and Woman's hospitals in Detroit and St. Joseph's Hospital in Ann Arbor. At the University Hospital, Ann Arbor, where he was Chairman of the Department of Ophthalmology, he established one of the first three-year progressive residency training programs in Ophthalmology, and this progressive training program has been a model for many others throughout the country to this day.

Outstanding among the many citations and honors bestowed upon Doctor Parker during his active years were an honorary Doctor of Science degree from the University of Pennsylvania in 1926 and another from the University of Michigan in 1935. He received the Knapp Medal from the Section on Ophthalmology of the American Medical Association in 1916 for his research on "The Relation of Tension of the Eyeball to Papilledema." He made numerous contributions to the ophthalmic literature, and at one time was a member of the editorial staff of the Archives of Ophthalmology.

Doctor Parker's military connections were high points of interest for him. He loved to recount his experiences as an Ensign in the U. S. Navy in 1898, during the Spanish-American War, when he served on the USS Yosemite. Between 1917 and 1919 he was Colonel in the Medical Corps, in charge of the Division of Head Surgery in the Office of the Surgeon General, Washington, D. C., during World War I.

Doctor Parker became a member of the Academy in 1903, when its membership numbered less than two hundred. He was keenly interested in its growth, and for many years took an active part in its educational program. Doctor Parker was President of the Academy in 1922.

Membership in other professional societies included the American Medical Association, of which he was Chairman of the Section on Ophthalmology in 1916; The American Ophthalmological Society, of which he was President in 1928; and French and British ophthalmological societies. He was also a Member of the Council of the International Federation of Ophthalmological Societies, and a Fellow of the American College of Surgeons. During his college years he was a member of the Delta Kappa Epsilon and the Nu Sigma Nu fraternities. He was an active participant in the Detroit City Club, the University Club of Detroit, the Detroit Country Club, Grosse Point Club, and the Yondotego Club.

For all those who knew Walter Parker, his passing has meant a deep personal loss. He exemplified the old school of professional men who maintained the bearing of a perfect gentleman at all times. He was a fine teacher, whose teachings were enriched by his keen interest in the Arts and by those qualities which marked him as a man of broad intellectual attainment.

In Memoriam

Richard Leslie Bower.....Carmel, California
March 26, 1955

Clyde Alvin Clapp.....Baltimore, Maryland
April 9, 1955

Harry David Earl.....Yucaipa, California
February 15, 1955

Bert Edward Ellis.....Indianapolis, Indiana
June 3, 1955

Sidney Saurin Evans.....Memphis, Tennessee
March 30, 1955

Kaufman Schlivek.....New York, New York
March 31, 1955

Harry Vernon Thomas.....Clarksburg, West Virginia
April 21, 1955

TRANSACTIONS

American Academy of Ophthalmology and Otolaryngology

Official Publication of the American Academy of Ophthalmology and Otolaryngology
Published under the direction of the Editorial Board
WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
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All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

CLINICS IN MEXICO

The Academy Instruction Courses will be extended to include Clinics in Mexico City at the conclusion of the five-day meeting of the annual convention in Chicago, October 9 to 14. The eye, ear, nose and throat clinics in several hospitals in the City of Mexico will be open to members of the Academy and their friends on Monday and Tuesday, October 17 and 18. Attendance is free and optional at any one or more of the city and federal hospitals. Transportation will be arranged. Advance registration for the clinic visits will not be required.

A survey of bookings for the Clinics in Mexico shows that already the country is represented from California to the Atlantic seaboard, and from Canada to the Gulf of Mexico. Sixteen of those now booked were members of the Bermuda Continuation Cruise last year.

Bookings for the Blue Ribbon Coach and for the Flagship are about equally divided. By the Coach flight there is a real dollar saving without a sacrifice in comfort, speed, or safety. Do not hesitate to go Coach if you wish to economize where it will hurt so little, if at all.

Ground arrangements and hotels in Mexico are the same in all respects for all members of the Clinic party whether they use Coach or Flagship.

How early a "waiting list" will develop no one can say. It seems to be on its way soon. Do not wait too long if you hope for membership in the Clinics in Mexico.

Write NOW to Leon V. Arnold, 33 Washington Square West, New York 11, N. Y., for full information and reservations. Your deposit should go to him, too.

Taxco and Cuernavaca

This two-day trip, Wednesday and Thursday, will be one of the loveliest, most interesting, and most varied experiences of your life. Here it is. On your return from this trip, I believe you will say, "If we had done nothing else during our time in Mexico, we've had our money's worth."

Taxco is a source of unending interest to historians, artists, photographers, and writers. In it lingers the charm of the sixteenth and seventeenth centuries, unchanged by time. The highway sweeps along the sides of the cliff. It passes under an ancient viaduct, makes a sharp turn and a breathtaking view of Taxco comes into view. A cobblestone road leads up a steep incline into the center of the picturesque town. The traditional plaza is flanked on one side by the famous Santa Prisca Church, on another

by the ancient homes and offices of former governors, and on others by shops displaying regional handicrafts, silver, leather goods, shoes and sandals.

The Santa Prisca Church dominates the town. It was built by a French miner, Jose de la Borda, as a lasting testimony of his gratitude to God for the wealth he obtained from the mine once located on the site. The Church is one of the finest examples of the Churriqueresque architecture in the world.

Everywhere throughout the town are winding, narrow streets, quaint nooks, sun-drenched terraces, colonial houses and entrancing spots. The Mexicans have recognized the importance of protecting Taxco's beauty and have decreed it a national monument and forbidden changes. Streets and lanes still are paved with cobblestones laid down by the town founders, and bridges are the same as those built by the first Spaniards who arrived.

Taxco is a paradise. Its beauty and charm, its quaint streets, it lovely homes perched high along the side of the mountain, its patios and balconies laden with flowers and overgrown with vines fill the city with enchanting views.

Cuernavaca, sheltered by wooded mountains and ideally located on the sun-bathed slopes of a lush semitropical valley, was once a quiet Indian community, the favorite resort of the reigning Aztec nobility. The natives defended the place fiercely when it was attacked by the Spaniards in 1521, but they lost. The place fell as a prize to Hernando Cortez. This was one of thirty such grants awarded to him by the Spanish crown, and it became a part of his vast estate. Enchanted by its location, he built a palatial home, and in 1529 he founded a Franciscan monastery, which later became the Cathedral and in which native craftsmen were permitted to incorporate many of their Aztec designs.

In 1716 Jose de la Borda, the French miner who amassed great wealth from his silver mines in Taxco, built a huge mansion in beautiful gardens, which we shall visit. Later this place was chosen for residence by the ill-fated Emperor Maximilian and the Empress Carlota.

Cuernavaca grew and prospered and became a state capital. Today, as in ancient times, it is a popular resort. Narrow streets, many still paved with cobblestones, and lovely old homes with balconies blanketed with bougainvillea and other vines connect the present with the centuries-old past. Here are the homes of aristocratic, diplomatic, and political Mexico.

Flanking one side of the main Plaza is the Palace of Cortez, which we shall visit to see the murals by Diego Rivera on one side of the veranda walls. Just off the Plaza are attractive shops.

While Taxco and Cuernavaca are the dazzling highlights of this tour we must not overlook the sparkling beauty of the smaller features.

San Angel, where flowers fall in bright cascades over mellow walls, is a lovely town surrounded by large orchards and contains many handsome country homes.

The Pedrigal is another aristocratic residential section.

Desert of Lions is not a desert and there are no lions. It is a forest of ancient trees, making a beautiful wooded national park through which we will drive. This is a popular holiday and picnic spot.

University City has only recently been completed. The new buildings, modern in both style and appointments, house all the departments of the University of Mexico.

Hacienda Hermosa is a charming resort created within the huge walled area of Cortez's Summer Palace. Later it was a monastery. You'll love it. You'll wonder how it could have been done. We lunch here.

Editorials

SPECIAL NOTICE

In past years the Alumni Dinners and the Evening of Diversion have been held on Monday evening, and the Banquet on Wednesday evening. This year the Alumni Dinners are scheduled for Wednesday evening and the Banquet for Monday evening. In lieu of the Evening of Diversion, following the Banquet on Monday evening the Academy will furnish top professional entertainment as well as an excellent dance orchestra which is guaranteed to entice even the most languid ones from their seats to the dance floor. Furthermore, there absolutely will be no speeches.

More detailed information about the arrangements will appear in subsequent issues of the TRANSACTIONS. Tables may be reserved in advance when desired and will be held for late arrivals. Pre-dinner and concurrent refreshments will be available at the table. What more can you ask?

DINNER FOR PLASTIC SURGEONS

The Commitee on Plastic and Reconstructive Surgery will sponsor a subscription dinner for surgeons interested in ophthalmologic, otolaryngologic and maxillofacial surgery at 6:30 p.m., Tuesday, October 11, 1955, in the Grand Ballroom of the Palmer House.

Following the dinner, a program of interest to ophthalmic surgeons will be held in Private Dining Room 14. Included in this program will be a motion picture on Exenteration, by Charles E. Iliff, M.D.,; a paper on Peritoneal Grafts for Conjunctival Replacement, by James Allen, M.D.; and a discussion of Basic Causes for Failure in Grafting Skin and Mucous Membrane of the Lids and Socket, by Milton Singer, M.D.

The program for otolaryngologic and maxillofacial surgeons, which will be held in the Crystal Room following the Dinner, will include a paper by Oscar J. Becker, M.D., on General Principles of the Corrective Nasal Plastic Operation; a paper on Principles of the Primary Repair of the Cleft Lip, by William C. Huffman, M.D.; a report on A Technique for Surgical Correction of Lop Ears, by John Marquis Converse, M.D., Anthony Nigro, M.D. (by invitation), Frederick A. Wilson, M.D. (by invitation), and Norman Johnson, M.D.; and a discussion of The Median Forehead Flap for the Repair of Defects of the Eyelids and Nose, by V. H. Kazanjian, M.D.

It is urged that reservations for this dinner be made in advance by sending your order with remittance to Dr. W. L. Benedict, Executive Secretary-Treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Price of ticket, $7.50.

NEW INSTRUMENTS

If you have a new instrument which you wish to report at the annual meeting of the Academy in October, please notify the Secretary in charge of your Scientific Section as soon as possible. The deadline is September 15. It will not be possible, as formerly, to make arrangements during the meeting for such presentations.

The Secretary for Ophthalmology is Dr. Kenneth L. Roper, 55 East Washington Street, Chicago 2. Dr. Lawrence R. Boies, E537 University Hospital, Minneapolis 14, is Secretary for Otolaryngology.

FINANCIAL REPORT

Annual Audit for Calendar Year — 1954

April 28, 1955

American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota

Gentlemen:

As requested, we have examined the balance sheet of the American Academy of Ophthalmology and Otolaryngology, an unincorporated association, Rochester, Minnesota, for the year ended December 31, 1954, and the related statements of operations and net worth for the year then ended. Our examination was made in accordance with generally accepted auditing standards and included such tests of the accounting records and such other auditing procedures as we deemed necessary in the circumstances. We submit herewith our report, which includes the following statements:

Exhibit

"A"—Balance Sheet, December 31, 1954
"B"—Statement of Operations, Year ended December 31, 1954
"C"—Analysis of Funds from Special Contributions
"D"—Analysis of Net Worth Account
"E"—Analysis of Increase in Market Value of Securities

Schedule

1—Securities owned, December 31, 1954

FINANCIAL CONDITION

A balance sheet as at December 31, 1954, appears in Exhibit "A." Subject to the comments contained in this report, we believe that this statement correctly reflects the financial condition of the American Academy of Ophthalmology and Otolaryngology on December 31, 1954. The following comments describe in more detail the nature of the various assets and liabilities and the extent of our verifications.

Current Assets, $23,396.41

Cash on deposit at the various banks, totaling $4,810.83, was verified by communicating with the depositories, the funds in the Canadian bank being stated in U. S. dollars. Petty cash fund of $25.00 was verified by actual count during the course of our examination.

Membership dues receivable, in the amount of $1,514.00, consists of delinquent dues for 1954 and prior years which were verified from the records only. Other receivables totaling $2,027.86, as taken from the records, consist of amounts due from members and others in the sum of $1,127.75 for films, slides, abstracts, manuals and monographs; $855.31 represents cash received in 1954 by the custodian of the Educational Fund and remitted to the Academy in 1955; and $44.80 due from Railway Express for shipment of periodicals lost in transit. The inventory of abstracts, manuals and monographs, Bound Volumes, films and slides, in the amount of $14,994.72, was taken from the inventory records as prepared by employees of the Academy without further verification except as to testing of prices, extensions and footings.

Investments, $217,642.49

In Schedule No. 1, we present a schedule of bonds and stocks which had a market value of $217,642.49 on December 31, 1954. U. S. Savings Series G Bonds are stated at maturity values rather than at current redemption prices.

Securities in the Educational Fund which are held for safekeeping by the Trust Department of the First National Bank, Minneapolis, Minnesota, were verified by communicating with the custodian of these securities. U. S. Savings Series G Bonds in the Wherry Memorial Fund are held by Dr. W. L. Benedict, Executive Secretary-Treasurer of the Academy, and were inspected by us. Interest earned on bonds since the dates on which last payments became due has not been accrued. An analysis of the increase in the market value of these investments during the year is shown in Exhibit "E."

Fixed Assets, $26,630.30

Furniture, fixtures and equipment amounting to $11,840.89 represents equipment acquired by the Academy over a period of years. Equipment acquired in years prior to September 1, 1942, which had been charged to operating expenses as purchased, was inventoried and appraised at a market value of $1,420.28 as of January 1, 1944, and set up on the books for that amount. Subsequent purchases of equipment have been verified through an examination of supporting vouchers, with the additions during the current year amounting to $1,387.42. Depreciation has been deducted and charged to operations at a rate that appears to be adequate.

Embryology of the Eye motion picture film and slides, in the amount of $40,900.00, represents the cost to complete a medical motion picture film and slides entitled "Embryology of the Eye," by Sturgis-Grant Productions,

Inc. Prints made from the master film are available for sale at approximate cost. The cost of the original film and slides is being amortized at a rate that appears to be adequate.

Deferred Charges, $110,803.37

Unexpired insurance premiums as computed by us amounted to $507.81.

Prepaid postage deposit of $204.80, verified by correspondence, represents the balance of cash advanced to the U. S. Post Office at Omaha, Nebraska, to be used as postage on subsequent mailings of the TRANSACTIONS and supplements.

Supplies in the amount of $90.75, as inventoried by employees without further verification by us, consists of lapel buttons which are awarded to Senior Fellows.

Payments totaling $90,000.01 have been made to Sturgis-Grant Productions, Inc., to produce a motion picture in color with sound, entitled "The Embryology of the Human Ear." Part I was completed in 1953 at a cost of $50,000.01. On December 8, 1953, a new agreement was negotiated to complete Parts II and III of the film on or before August 31, 1954, at an additional cost of $40,000.00. This project was completed in the subsequent year with some additional costs which are not included in the above amounts or shown as a liability on the balance sheet.

Payments totaling $20,000.00 have been made to the National Academy of Sciences, acting as fiscal agent for the American Registries of Pathology, for the printing of 3,000 copies of the forthcoming publication entitled "Atlas of Otolaryngic Pathology." The total cost of the project will be approximately $40,740.00. The Academy will be reimbursed, in part or in full, when funds become available from sales of the "Atlas of Otolaryngic Pathology," for which the American Registries of Pathology will be the distributor.

Current Liabilities, $52,469.64

A note payable in the amount of $40,000.00, due March 22, 1955, to the First National Bank, Rochester, Minnesota, was verified by correspondence.

Accrued expenses of $8,862.79 represents unpaid December 1954 printing costs of the TRANSACTIONS and other current expenses. Accrued social security and withholding taxes due as of December 31, 1954, amounted to $652.40.

Prepayment for Bound Volumes, etc., totaling $2,749.45, consists of payments made for Bound Volumes, etc., to be delivered in 1955.

Deferred income, membership dues, totaling $205.00, represents dues paid in advance by Academy members.

Undisbursed Funds from Special Contributions, $5,407.72

Undisbursed funds from contributions received and designated for "Specific Projects" are analyzed in Exhibit "C," which shows 1954 transactions as well as prior year balances.

Net Worth, $320,595.21

An analysis of the net worth of the Academy is shown in Exhibit "D." The net worth increased from $290,065.00 on December 31, 1953, to $320,595.21 on December 31, 1954, as a result of an increase in market values of securities totaling $15,189.24, and a net gain for the year in the amount of $15,340.97.

OPERATIONS

A statement of operations for the year ended December 31, 1954, is presented in Exhibit "B." A summary and a comparison with the results for the year 1953 is as follows:

	Year 1954	*Year* 1953	*Increase*
Revenues	$250,533.62	$200,018.18	$50,515.44
Operating Expenses	235,974.40	196,331.32	39,643.08
Operating Income	$ 14,559.22	$ 3,686.86	$10,872.36

Revenues from all sources totaled $250,533.62 for the year ended December 31, 1954, as compared to $200,018.18 for the year ended December 31, 1953, shows an increase of $50,515.44. Operating expenses totaled $235,974.40 for the year ended December 31, 1954, as compared to $196,331.32 for the prior year.

GENERAL

Upon examination of the insurance policies in force, we found fire and extended coverage on furniture, fixtures and equipment in the amount of $7,500.00 with 80% co-insurance. Paper stock, publications, etc., in custody of the printer at Omaha, Nebraska, are insured for $12,000.00 with 80% co-insurance. Comprehensive general liability insurance is in force, covering bodily injury in the amount of $25,000.00 for each person and $25,000.00 for each accident, and this coverage is increased to special limits of $50,000.00 each person and $100,000.00 each accident at the coming annual convention at the Palmer House, Chicago, Illinois.

Property damage liability is covered in the amount of $5,000.00 for each accident with $25,000.00 aggregate operations, $25,000.00 aggregate protective and $25,000.00 aggregate contractural.

Workmen's compensation insurance, which is being carried at the statutory amounts, includes coverage for all Academy employees,

including additional help hired at the annual conventions.

The Executive Secretary-Treasurer of the Academy is bonded for $25,000.00, while the principal office employees are each bonded for only $1,000.00, with the exception of the manuscript editor who is bonded for $2,500.00. In view of large amounts of cash handled at conventions by office employees, their fidelity position bond coverage, in our opinion, is inadequate.

The loss of money, securities and other property is covered up to $2,400.00 within the premises and $2,100.00 outside the premises, against safe burglary and messenger robbery. At the annual convention the coverage for loss within the premises on money, securities and other property is increased to $40,000.00, with a limit of $20,000.00 on money.

We acknowledge the very courteous assistance extended to us by Dr. W. L. Benedict, Executive Secretary-Treasurer of the Academy, and his staff during the progress of this examination.

OPINION

In our opinion, subject to the comments in the preceding paragraphs, the accompanying balance sheet and related statements of operations and net worth present fairly the financial position of the American Academy of Ophthalmology and Otolaryngology at December 31, 1954, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,

BYERS, WOBSCHALL & MILLER
Certified Public Accountants
By Edw. A. Wobschall, C.P.A.

EXHIBIT "A"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Balance Sheet as at December 31, 1954

ASSETS

Current Assets:			
Cash, First National Bank, Rochester, Minnesota		$ 1,501.99	
Cash, First National Bank, Minneapolis, Minnesota		537.01	
Cash, Bank of Montreal, Toronto, Canada		2,771.83	
Petty cash fund		25.00	
Membership dues receivable, delinquent		1,514.00	
Accounts receivable, others		2,027.86	
Checks held for collection		24.00	
Inventory: abstracts, manuals, monographs, films, etc.		14,994.72	
Total current assets			$ 23,396.41
Investments: (Schedule No. 1)			
Bonds and stocks			217,642.49
Fixed Assets:			
Furniture and fixtures	$11,840.89		
Less reserve for depreciation	6,080.59	5,760.30	
Embryology of the Eye: Film & Slides	40,900.00		
Less reserve for depreciation	20,030.00	20,870.00	26,630.30
Deferred Charges:			
Prepaid insurance		507.81	
Prepaid postage		204.80	
Supplies		90.75	
Embryology of the Human Ear: Motion Picture Film		90,000.01	
Atlas of Otolaryngic Pathology		20,000.00	110,803.37
Total Assets			$378,472.57

LIABILITIES AND NET WORTH

Current Liabilities:		
Notes payable	$ 40,000.00	
Accrued payroll taxes	652.40	
Accrued expenses	8,862.79	
Prepayment for Bound Volumes, etc.	2,749.45	
Deferred income, membership dues	205.00	
Total current liabilities		$ 52,469.64
Undisbursed Funds from Special Contributions: (Exhibit "C")		
Wherry Memorial Fund		5,407.72
Net Worth (Exhibit "D")		320,595.21
Total Liabilities and Net Worth *		$378,472.57

* Subject to report comments

EXHIBIT "B"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Operations
Year Ended December 31, 1954

Revenues:			
Membership dues			$ 64,780.00
Membership applications			4,698.00
Abstracts, manuals and monographs			8,777.76
Certificates			1,395.50
Conservation of hearing			250.00
Films and slides			2,925.00
Testing and calibration of tonometers			1,428.00
Royalties: Atlas and Textbook of Ophthalmic Pathology			1,795.80
Convention			85,446.00
Home Study Courses			7,310.00
TRANSACTIONS			64,034.75
Income from securities:			
Interest, Educational Fund		$ 3,997.50	
Dividends, Educational Fund		2,419.06	6,416.56
Other income			1,276.25
Total Revenues			250,533.62
Operating Expenses:			
General administrative		79,645.63	
TRANSACTIONS		68,237.03	
Home Study Courses		8,695.80	
Convention expenses		74,950.75	
Educational Fund:			
Trustee fees	$ 250.19		
Depreciation, film and slides	4,195.00	4,445.19	
Total Operating Expenses			235,974.40
Operating Income			14,559.22
Other Income:			
Gain on sale of securities		721.75	
Gain on sale of equipment		60.00	781.75
Net Gain			$ 15,340.97

EXHIBIT "C"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Funds from Special Contributions

WHERRY MEMORIAL FUND	
Balance, December 31, 1953	$ 5,292.72
Receipts:	
Interest on U. S. Savings Bonds, year 1954	115.00
Balance, December 31, 1954	$ 5,407.72
SUB-COMMITTEE ON NOISE IN INDUSTRY FUND	
Balance, December 31, 1953	$ 4,479.81
Receipts:	
Contributions received from outside sources	67,697.03
Total	72,176.84
Less: Transfers to Sub-Committee on Noise in Industry (Los Angeles, California) *	72,176.84
Balance, December 31, 1954	None

* This amount received by the Sub-Committee on Noise in Industry as shown in audit report submitted to the Academy.

EXHIBIT "D"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Net Worth Account For Year Ended December 31, 1954

Net Worth, December 31, 1953		$290,065.00
Additions:		
Increase in market value of securities during 1954 (Exhibit "E")	$ 15,189.24	
Net gain, year ended December 31, 1954 (Exhibit "B")	15,340.97	30,530.21
Net Worth, December 31, 1954		$320,595.21

EXHIBIT "E"

Analysis of Increase in Market Value of Securities

	Cost	*Market Value*	*Increase* **Decrease**
December 31, 1953	$218,230.21	$226,376.25	$ 8,146.04
December 31, 1954	189,707.21	213,042.49	23,335.28
Difference	**$28,523.00**	**$13,333.76**	$15,189.24

SCHEDULE No. 1

American Academy of Ophthalmology and Otolaryngology
Securities Owned, December 31, 1954

EDUCATIONAL FUND

BONDS	RATE %	MATURITY	PAR VALUE	COST		MARKET VALUE *
Citizens Building Co. of Cleveland, 1st Leasehold Sinking Fund	6	1/ 1/46	$ 5,000.00	$ 5,000.00	(1)	None
Van Swerigen Co. Certificates of Indebtedness, Series 1925	6	12/31/48	6,000.00	3,477.03		$ 1,200.00
Dominion of Canada, 8th Victory Loan Series P5	3	10/ 1/63	7,500.00	7,612.50	(2)	7,846.88
Dominion of Canada, 9th Victory Loan Series P7	3	9/ 1/66	2,000.00	1,880.00	(2)	2,072.50
U.S.A. Savings, Series F	—	12/ 1/55	13,500.00	9,990.00		12,987.00
U.S.A. Savings, Series G	2½	3/ 1/56	6,000.00	6,000.00		6,000.00
U.S.A. Savings, Series G	2½	5/ 1/56	2,000.00	2,000.00		2,000.00
U.S.A. Savings, Series G	2½	11/ 1/56	25,000.00	25,000.00		25,000.00
U.S.A. Savings, Series G	2½	7/ 1/58	30,000.00	30,000.00		30,000.00
U.S.A. Savings, Series G	2½	12/ 1/58	25,000.00	25,000.00		25,000.00
U.S.A. Savings, Series G	2½	3/ 1/59	15,000.00	15,000.00		15,000.00
U.S.A. Savings, Series G	2½	7/ 1/60	30,000.00	30,000.00		30,000.00
Total Bonds				$160,959.53		$157,106.38

STOCKS	KIND	PAR VALUE	SHARES			
Citizens Building Co. of Cleveland	Shares	No Par	50	None		None
Commonwealth Edison Co.	Common	$25.00	100	$ 2,619.00		$ 4,587.50
E. I. DuPont De Nemours & Co. ...	Common	5.00	60	2,347.31		10,050.00
Louisville Gas & Electric Co.	5% Preferred	25.00	200	5,450.00		6,100.00
Massachusetts Investors Trust	Trust Cert.	1.00	1,251	18,254.37		35,015.49
Ophthalmic Publishing Co.	Capital	20.00	5	None		100.00
Northern Illinois Gas Co.	Common	5.00	4	77.00		83.12
Total Stocks				$ 28,747.68		$ 55,936.11
TOTAL EDUCATIONAL FUND (3)				$189,707.21		$213,042.49

WHERRY MEMORIAL FUND	RATE	MATURITY	PAR VALUE			
U.S.A. Savings, Series G (4)	2½%	1955	$4,600.00	$ 4,600.00		$ 4,600.00
TOTAL INVESTMENTS ...				$194,307.21		$217,642.49

* Except for U.S.A. Savings, Series G Bonds, which are included at maturity values
(1) No market available
(2) U.S. dollars
(3) Held for safekeeping by Trust Department, First National Bank, Minneapolis, Minnesota
(4) Held by Dr. W. L. Benedict, Executive Secretary-Treasurer, Rochester, Minnesota

HOME STUDY COURSES

Secretary:
Daniel Snydacker

Associate Secretary:
Ben H. Senturia

Ophthalmology

MONTH	SUBJECT	FACULTY
September	Embryology, Anatomy and Neuro-anatomy	Raymond E. Meek John W. Henderson
October	Physiology	Irving H. Leopold Robison D. Harley
November	Optics and Principles of Biomicroscopy	Arthur Linksz Edwin F. Tait
December	Refraction	Frank W. Newell Manuel L. Stillerman
January	External Diseases, Bacteriology and Virology	James H. Allen S. Rodman Irvine Samuel N. Key, Jr.
February	Perimetry and Neuro-ophthalmology	P. J. Leinfelder Cecil W. Lepard
March	Extraocular Muscles	Hunter H. Romaine Hermann M. Burian
April	Ophthalmoscopy and Medical Ophthalmology	John P. Macnie Wm. Banks Anderson Frank C. Winter
May	Pathology	Michael J. Hogan John S. McGavic
June	Pharmacology and Therapeutics	Kenneth C. Swan Alfred J. Kreft

Otolaryngology

September October	Anatomy I Anatomy II	{Maurice F. Snitman {Peter N. Pastore
November	Histology	Leland G. Hunnicutt Frederic J. Pollock
December	Pathology	George F. Reed
January	Physiology	William C. Thornell Harold F. Schuknecht
February	Bacteriology	Henry L. Williams James W. McLaurin
March	Pharmacology	Clifford F. Lake G. O'Neil Proud
April	Hearing Function and Testing	Arthur L. Juers Victor Goodhill
May	Vestibular Function and Testing	William J. McNally John R. Lindsay
June	Otological Acoustics	Scott N. Reger Clair M. Kos

Application forms for 1955-1956 may be secured from the Executive Secretary-Treasurer of the Academy, Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minnesota. Registrations must be completed before August 15. The fee is $20.00.

HISTORY OF THE INTERNATIONAL ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

On October 11, 1939, during the annual session of the American Academy of Ophthalmology and Otolaryngology, thirty-two secretaries of various widely scattered eye, ear, nose and throat societies met at a luncheon at the Palmer House, Chicago, discussed the advantages and opportunities of such meetings, and perfected a permanent organization of eye, ear, nose and throat society secretaries. Officers and a Council for the ensuing year were selected, and a petition asking the support and sponsorship of the American Academy of Ophthalmology and Otolaryngology was drawn up for presentation to the Council of the Academy.

Thus was born the American Association of Eye, Ear, Nose and Throat Society Secretaries. This organization was the brain child of Dr. William P. Wherry, then Executive Secretary of the American Academy of Ophthalmology and Otolaryngology. Doctor Wherry had long foreseen the potential strength of an organized group of society secretaries, nurtured under the wing of the Academy, with a program of activities streamlined to render needed service both to its component societies and to its Academy godfather. Doctor Wherry fostered and promoted the idea in the pages of the Academy *Bulletin* and was instrumental in bringing the charter members together for the organizational meeting.

During the following year, an announcement regarding the organization and purposes of the new society of secretaries was sent to 81 other secretaries of eye, ear, nose and throat societies—local, state and national. The response was very gratifying. About one half of the number answered the letter, heartily approving of the idea. Many asked for immediate aid in solving certain problems confronting them and their societies. Most of the queries received were much alike; they dealt with such problems as types of programs, procedure at meetings, organizational problems, and how to provide attractive meetings in order to secure good attendance. These problems seemed to be universal and common to all secretaries, indicating the need for such an organization and for a common clearing house of information and help for its members. The questions were sent to the officers and directors of the new society, and the answers were published in the *Bulletin* of the American Academy of Ophthalmology and Otolaryngology, August 1940. Personal letters containing the information desired were sent to those secretaries who were not members of the Academy and who therefore did not have access to the *Bulletin*.

The second meeting of the Association was held on October 9, 1940, during the session of the Academy at Cleveland. Luncheon was again provided through the courtesy of the Academy. Inspiring talks upon the aims of the society and its potentialities for fine achievement were made by Dr. Frank R. Spencer, Boulder, Colorado, President-Elect of the Academy, by Dr. John L. Myers, Kansas City, Missouri, and Dr. William L. Benedict, Rochester, Minnesota, Secretaries, respectively, for Otolaryngology and Ophthalmology in the Academy, by Dr. Walter H. Theobald, Chicago, President of the Association, and by others. Further progress was made in establishing a more solid business foundation for the Association, and an active discussion was held regarding

DR. WILLIAM P. WHERRY

ways and means of dealing with certain questions and matters of interest common to the individual members.

As its membership became more closely knit, the new society of secretaries continued to grow and flourish, both in size and in the promotion of its various projects. Annual meetings were held during the conventions of the American Academy, always at a dinner of banquet proportions furnished through the courtesy of the Academy. Inspiring programs were given by prominent speakers, and many valuable hints and suggestions gleaned from these talks and from the general discussion which followed were taken home by the attending secretaries, to be applied toward improvement in their own efficiency and in the functioning of their societies. Certain projects and activities designed to render mutual aid in the interim between the annual meetings were adopted and put into effect. Printed programs were interchanged among the secretaries of each geographical district and between neighboring districts, information was relayed back and forth regarding especially good programs, and a clearing house was maintained at the central headquarters for the reception and dissemination of information helpful in solving the many problems faced by a secretary. Numerous inquiries were received regarding such matters as program planning, guest speakers versus "home talent," how to handle the business affairs of a society, how to organize a new society, suggestions for a workable constitution and by-laws, and how to stimulate better attendance at meetings. In many instances the replies to these questions were helpful and timely, and were greatly appreciated.

As the Association continued to develop and to expand its scope of activity, it was found that a code of regulations was needed for guidance in the administration of its affairs. A committee composed of Dr. Sam H. Sanders of Memphis, Dr. W. E. Keith of Kansas City and Dr. Kenneth L. Craft of Indianapolis met in St. Louis in January 1944 and drafted a tentative Constitution and By-laws. Copies were sent to all the members, some changes for improvement were made, and the document was ratified and adopted at the 1944 meeting. Recent broadening of the Association's activities has made it advisable to change the original Constitution and By-laws in certain respects. This revision is now in process.

A factor which greatly facilitated the work of the Association was the change to a more favorable time for holding the annual meeting. For the first few years the meetings had been held at a noon luncheon sandwiched in between the morning and afternoon sessions of the Academy. This left little time for business matters, for the formal program, or for general discussion by the attending members. A flood of letters requesting a change in meeting time descended upon the office of the Executive Secretary-Treasurer of the Academy, Dr. William L. Benedict, and since 1944 the annual meeting has been held in the evening at a dinner furnished through the generosity of the Academy. Another courtesy granted by the Academy which has been

of great help was permission to use the Academy TRANSACTIONS for the publication of the minutes of the meetings and the dissemination of information and news of interest to society secretaries.

In 1944 a training course for secretaries was organized following the receipt of many requests for instruction which would help secretaries to render better service to their societies. The first course was presented at a luncheon and was attended by twenty-five secretaries. The response was so enthusiastic and the demands for repetition so numerous that the course was presented the following year at the regular meeting. At the 1954 meeting several of those present suggested that the course be repeated in the near future. This can be done if enough members are interested.

The organization of the Pan-American Congresses of Ophthalmology and of Otolaryngology stimulated plans for extension of Association activities to Central and South America. Dr. Moacyr E. Alvaro of Sao Paulo, Brazil, Chairman of the Department of Ophthalmology at the University of Sao Paulo, joined the Association in 1944 and became one of its most enthusiastic members. District 9, which includes the Latin-American societies, was added, with Doctor Alvaro as Director, a position which he still holds. At the cost of much personal sacrifice and a tremendous amount of promotional work, the societies in his district were made aware of the American Association of Eye, Ear, Nose and Throat Society Secretaries and of its functionings and policies. To date, as a result of Doctor Alvaro's fine missionary work, fifteen Latin-American societies now are included in his district.

In 1946 twenty-one EENT specialists from South American countries came to Chicago to attend the first Pan-American Congress of Otolaryngology. These men were guests at the meeting of the Association. As they were introduced, many of them gave short talks upon ways and means of improving Pan-American relations, advocating closer collaboration between the EENT societies of Latin-America, Canada and the United States and stressing the advantages and opportunities afforded by such an organization as the Association of Secretaries. It was agreed that in addition to the professional advantages to be gained through the meetings and interchange of ideas, the workings of such a group should provide an unusually fine opportunity for development of the Good Neighbor Policy between nations. As Director-General of the entire Central American and South American district, Doctor Alvaro continues the organizational program among the various societies in these countries.

A word of appreciation for the generous and constructive assistance given this society by the American Academy should be noted. Conceived in the mind of the late Dr. William P. Wherry, the Secretaries' Association has received continuous and unstinting support from Dr. William L. Benedict, who in 1942 succeeded Doctor Wherry as Executive Secretary-Treasurer, from Dr. Dean M. Lierle, Secretary for Instruction in Otolaryngology and Maxillofacial Surgery, from Dr. A. D. Ruedemann, Secretary for Instruction in Ophthalmology, and from the Council and other officers of the Academy. Without their help and advice this organization could not have developed to its present stature. In return, the Secretaries' Association has tried to express its appreciation by cooperating heartily with the Academy in the promotion of various projects and in every way possible.

Perhaps the most ambitious program of this type undertaken by the Association was the critical survey, made an-

nually over a period of several years, of the various Academy Instructional Courses. This investigation covered practically every course and each instructor upon the program. A voluminous amount of information and constructive criticism was collected and turned over to the Academy officials. As a result there has been a marked improvement in the quality and the scope of the instructional program. This was a tremendous undertaking, but the society was more than happy to do it.

The accomplishment of these various projects gave the Secretaries' Association confidence in its strength and capabilities and an awareness of its potentialities. It began to develop "growing pains." At the meeting in 1951, President-Elect Moacyr Alvaro, who had so successfully promoted the aims and policies of the Association in Central and South America, proposed extension of these activities among European and Asiatic countries. The idea was received with enthusiasm, and a committee headed by Doctor Alvaro was appointed to contact the various EENT societies in these countries regarding the formation of a world-wide organization whose members could work and plan together for mutual benefit in solving problems common to all.

At the 1952 meeting, Doctor Alvaro, then President, reported that as a result of the committee's efforts, many societies all over the world had written to him expressing their desire to take an active part in an international group of eye, ear, nose and throat society secretaries. The general feeling was that "a great amount of international good will would be engendered through the workings of such an organization and that many benefits would accrue to each local society by learning what similar groups were doing to promote better educational meetings for eye, ear, nose and throat societies." At that time the name of the Association was changed from the American Association of Eye, Ear, Nose and Throat Society Secretaries to the International Association of Eye, Ear, Nose and Throat Society Secretaries.

Further reports upon the development of the "Alvaro plan" for extension were made at the annual meeting in 1953 by President James J. Monahan and Doctor Alvaro. President Monahan told of the progress which was being made by the Association in its contacts with various ophthalmological and otolaryngological societies in Europe, Asia and Africa. He mentioned various ways in which members of the International Association could help each other, both in meetings and by correspondence, and announced his plans for publication during the coming year of a definite program of Association activities for the furtherance of the international project. Doctor Alvaro reported that in his promotional work he had been in communication with secretaries of EENT societies all over the world and that many of them were very enthusiastic about the plan and requested that the work be continued.

As a means of facilitating the program of expansion the following resolutions were adopted at this meeting:

WHEREAS, there exists a need for increasing rapprochement among Ophthalmologists throughout the World; and

WHEREAS, there exists a need for increasing rapprochement among Otorhinolaryngologists throughout the World; and

WHEREAS, the International Association of Eye, Ear, Nose and Throat Society Secretaries finds itself in a strategic position to provide an exchange of ideas and opportunities;

NOW THEREFORE BE IT RESOLVED that the International Association of Eye, Ear, Nose and Throat Society Secretaries empower its Executive Council to establish a Bulletin at such a time and in such a form as may be practical and as it deems advisable; and

BE IT FURTHER RESOLVED that the Executive Council of the International Association of Eye, Ear, Nose and Throat Society Secretaries present this resolution to the Council of the American Academy of Ophthalmology and Otolaryngology during this annual session for approval and advice in its implementation; and

BE IT FURTHER RESOLVED that the Executive Council of the International Association of Eye, Ear, Nose and Throat Society Secretaries be requested to report its activities in relation to this resolution at the next annual meeting of this Association to be held at the Waldorf-Astoria in New York.

The program of international expansion of the Secretaries' Association is rapidly developing. The movement has spread into many lands across the seas. Leading specialists in ophthalmology and otolaryngology in these countries have been contacted and have expressed great enthusiasm for the project and a desire to help promote the program in their own communities. In a number of countries, skeleton organizations of new EENT societies have already been perfected. The international aspect of this movement is emphasized by representation upon the Directorate of the Association from the following countries: Argentina, Brazil, Canada, England, Egypt, France, Holland, India, Lebanon, New Zealand, Norway, Portugal, Puerto Rico. To expedite the program further, the news bulletin provided for in the above resolution has been established and is now being circulated both locally and abroad. Dr. Paul C. Craig of Reading, Pennsylvania, an outstanding leader and Past-President of the Association, is the Editor.

Many of our foreign colleagues have sent cordial invitations for all EENT doctors to attend their meetings and to visit them personally whenever possible. It is hoped that while traveling abroad, many will take advantage of this opportunity to promote cordial and harmonious international relations and to further this project of a world-wide organization of societies and individuals whose interests and problems are mutual. (Information regarding meetings abroad may be secured from Dr. Daniel S. DeStio, Association Secretary, 1006 Highland Building, Pittsburgh, Pa.)

Here in the United States, it was the pleasure and the privilege of the Association to meet and entertain many visitors from overseas who came to attend the meeting of the XVII International Congress of Ophthalmology held in conjunction with the meeting of the American Academy of Ophthalmology and Otolaryngology in New York in September 1954. Members of the Secretaries' Association and their wives served as special hosts to the visitors during the entire six days of the meeting. New friendships were made and good fellowship was engendered, both in the Foreign Delegates' Lounge and at the annual dinner meeting of the Association.

And so a small group of secretaries of eye, ear, nose and throat societies, mostly entire strangers to each other, drawn together at a luncheon fifteen years ago through the vision and foresight of Dr. William P. Wherry, has gradually developed into a strong closely knit organization whose influence and ideals for improvement in the functionings of EENT societies, for aid and instruction to secretaries, and for betterment of international relations generally, now encircle the globe. It is to be regretted that Bill Wherry could not have lived to see the growth of this society to heights and proportions no doubt far beyond his early hopes and expectations. He has had many honors bestowed upon him, both before his untimely demise and posthumously. Let us dedicate the present and future enterprises of the International Association of Eye, Ear, Nose

and Throat Society Secretaries to the honored memory of this man to whose vision and whose remarkable ability for accomplishment of purpose this organization owes its inception, its growth, and the unique position which it occupies today.

KENNETH L. CRAFT, M.D.

OFFICERS AND DIRECTORS OF THE INTERNATIONAL ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES WHO HAVE SERVED SINCE ITS ORGANIZATION IN 1939 AS THE AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

1939—Acting

President: Walter H. Theobald
Chicago, Ill.

Secretary: Kenneth L. Craft
Indianapolis, Ind.

1940

President: Walter H. Theobald

Secretary-Treasurer: Kenneth L. Craft

Directors:

William B. Clark
New Orleans, La.
Paul M. Moore
Cleveland, Ohio
W. Howard Morrison
Omaha, Neb.
John R. Myers
Syracuse, N. Y.
L. A. Schall
Boston, Mass.

1941

President: Walter H. Theobald

Secretary-Treasurer: Kenneth L. Craft

Directors:

Brittain F. Payne
New York, N. Y.
Lester A. Brown
Atlanta, Ga.
J. M. Carter
Detroit, Mich.
W. Howard Morrison
Mercer G. Lynch
New Orleans, La.
John R. Myers
J. C. Ogden
Long Beach, Calif.
R. T. Rank,
Milwaukee, Wis.

1942

President: Walter H. Theobald

Secretary-Treasurer: Kenneth L. Craft

Directors:

Brittain F. Payne
Lester A. Brown
Mercer G. Lynch
John R. Myers
J. C. Ogden
R. T. Rank
H. C. Thompson
Columbus, Ohio
W. E. Keith
Kansas City, Mo.

1943

President: W. Howard Morrison
Omaha, Neb.

Secretary-Treasurer: Kenneth L. Craft

Directors:

R. Townley Paton,
New York, N. Y.
Sheldon B. Freeman
Buffalo, N. Y.
Lester A. Brown
J. C. Ogden
Sam H. Sanders
Memphis, Tenn.
Daniel S. DeStio
Pittsburgh, Pa.
R. T. Rank
W. E. Keith

1944

President: W. Howard Morrison

Secretary-Treasurer: Kenneth L. Craft

Directors:

Burton E. Lovesey
Boston, Mass.
Sheldon B. Freeman
Lester A. Brown
Sam H. Sanders
Daniel S. DeStio
Vernon Leech
Chicago, Ill.
W. E. Keith
J. C. Ogden

1945—No meeting due to lack of transportation facilities during World War II. All Officers and Directors held over.

1946

President: Paul C. Craig
Reading, Pa.

Secretary-Treasurer: Kenneth L. Craft

Directors:

Burton E. Lovesey
Sheldon B. Freeman
Lester A. Brown
Sam H. Sanders
Daniel S. DeStio
Vernon Leech
W. E. Keith
J. C. Ogden

1947
President: Sam H. Sanders
Memphis, Tenn.
Secretary-Treasurer: Kenneth L. Craft
Directors:
Burton E. Lovesey
Sheldon B. Freeman
Lester A. Brown
Edley F. Jones
Vicksburg, Miss.
Daniel S. DeStio
William F. Mann
Chicago, Ill.
F. D. Hurd
Great Falls, Mont.
Barton E. Peden
Seattle, Wash.
Moacyr E. Alvaro
Sao Paulo, Brazil

1948
President: W. E. Keith
Kansas City, Mo.
Secretary-Treasurer: Kenneth L. Craft
Directors:
Burton E. Lovesey
James J. Monahan
Shenandoah, Pa.
Lester A. Brown
A. L. Peter
Akron, Ohio
Edley F. Jones
L. J. Lawson
Chicago, Ill.
F. D. Hurd
Barton E. Peden
Moacyr E. Alvaro

1949
President: Daniel S. DeStio
Pittsburgh, Pa.
Secretary-Treasurer: Kenneth L. Craft
Directors:
Burton E. Lovesey
James J. Monahan
Roderick Macdonald
Rock Hill, S. C.
A. L. Peter
Edley F. Jones
L. J. Lawson
F. D. Hurd
Barton E. Peden
Moacyr E. Alvaro

1950
President: Lester A. Brown
Atlanta, Ga.
Secretary-Treasurer: Daniel S. DeStio
Directors:
Burton E. Lovesey
James J. Monahan
Roderick Macdonald
A. L. Peter
Edley F. Jones
L. J. Lawson
F. D. Hurd
H. L. Goss
Seattle, Wash.
Moacyr E. Alvaro

1951
President: Kenneth L. Craft
Secretary-Treasurer: Daniel S. DeStio
Directors:
Burton E. Lovesey
James J. Monahan
Roderick Macdonald
A. L. Peter
Edley F. Jones
L. J. Lawson
F. D. Hurd
H. L. Goss
Moacyr E. Alvaro

1952
President: Moacyr E. Alvaro
Sao Paulo, Brazil
Secretary-Treasurer: Daniel S. DeStio
Directors:
Burton E. Lovesey
James J. Monahan
Roderick Macdonald
A. L. Peter
Edley F. Jones
L. J. Lawson
F. D. Hurd
H. L. Goss
Moacyr E. Alvaro

1953
President: James J. Monahan
Shenandoah, Pa.
Secretary-Treasurer: Daniel S. DeStio
Directors:
Burton E. Lovesey
James J. Monahan
Roderick Macdonald
A. L. Peter
Edley F. Jones
L. J. Lawson
F. D. Hurd
H. L. Goss
Moacyr E. Alvaro

1954
President: Edley F. Jones
Vicksburg, Miss.
Secretary-Treasurer: Daniel S. DeStio
Directors:
Burton E. Lovesey
James J. Monahan
Frederick C. Reel
Charleston, W. Va.
Lewis E. Morrison
Indianapolis, Ind.

Edley F. Jones
L. J. Lawson
F. D. Hurd
H. L. Goss
Moacyr E. Alvaro
Ian Fraser
Shrewsbury, England
Charles Thomas
Nancy, France
T. A. Vos
Holland
Johan C. Holst
Oslo, Norway
Henrique Moutinho
Lisbon, Portugal
S. N. Cooper
Bombay, India
M. Khalil
Cairo, Egypt
W. J. Hope-Robertson
Wellington, New Zealand
Dr. Diab
Beirut, Lebanon

1955

President: Roderick Macdonald
Rock Hill, S. C.
President-Elect: Willard F. Goff
Seattle, Wash.
Secretary-Treasurer: Daniel S. DeStio
Editor: Paul C. Craig
Directors:
Burton E. Lovesey
Benjamin F. Souders
Reading, Pa.
Frederick C. Reel
Roland H. Myers
Memphis, Tenn.
Lewis E. Morrison
Indianapolis, Ind.
Thomas S. Allen
Chicago, Ill.
A. E. Cruthirds
Phoenix, Ariz.
Willard F. Goff
Seattle, Wash.
Moacyr E. Alvaro
A. Urrets-Zavalia, Jr.
Cordoba, Argentina
Ricardo Fernandez
San Juan, Puerto Rico
Rene Charbonneau
Montreal, Que., Canada
Ian Fraser
Charles Thomas
T. A. Vos
Johan C. Holst
Henrique Moutinho
S. N. Cooper
M. Khalil
W. J. Hope-Robertson
Dr. Diab
Ernest Engelking
Heidelberg, Germany
Arych Feigenbaum
Tel Aviv, Israel
T. Nishihata
Tokyo, Japan

INTERNATIONAL ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES MEMBERS AND GUESTS PRESENT AT ANNUAL MEETING, 1954

James H. Allen, Louisiana
Thomas D. Allen, Chicago
M. E. Alvaro, Argentina
Raul Arganaraz, Argentina
James H. Beaumont, New Zealand
William L. Benedict, Minnesota
Carlos V. Charlin, Chile
A. A. Cinotti, New Jersey
Francis P. C. Claffy, Australia
A. C. Copper, The Netherlands
Kenneth Craft, Indiana
Paul C. Craig, Pennsylvania
A. E. Cruthirds, Arizona
Daniel S. DeStio, Pennsylvania
Arthur H. Downing, Iowa
A. Franceschetti, Switzerland
Adrien Fritz, Belgium
Frank C. Furlong, New York
N. I. Gershon, Georgia
E. I. Glenister, Canada
Harry N. Glick, Missouri
Willard F. Goff, Washington
Ernest E. Hessing, California
Francisco Martinez Hinojosa, Mexico
Paul H. Holinger, Illinois
William Hulse, Ohio
Charles E. Jaeckle, New Jersey
Francisco Jimenez, Mexico
Edley H. Jones, Mississippi
E. T. Juler, Ohio
Gosta Karpe, Sweden
Birgitta Knape, Finland
M. Mustafa Lahry, Egypt
Amos Leibel, Israel
Jesse M. Levitt, New York
Robert G. Linton, Australia
Roderick Macdonald, South Carolina
W. A. Manschot, The Netherlands
Antonio Queiroz Marinho, Portugal
Gatlin Mitchell, Texas
James J. Monahan, Pennsylvania
Albert F. Moriconi, New Jersey
Lewis E. Morrison, Indiana
Roland H. Myers, Tennessee
Miguel L. Olivares, Chile
Elizabeth Olmsted, New York

James H. Parker, Jr., Pennsylvania
Guillermo Pico, Puerto Rico
E. Lopez Quinones, Mexico
Julio C. Raffo, Peru
Hadi Rais, Tunisia
Aida Rifaat, Egypt
Sam H. Sanders, Tennessee
Tutomu Sato, Japan
Henry M. Scheer, New York
Benjamin Sheppard, Virginia
William Shokan, Texas
Thakur P. Singh, India
George A. Sisson, New York
Albert Steiner, Maryland
J. C. Strong, Jr., Colorado
Charles T. Sullivan, New York
Nicholas P. Tantillo, New York
Charles Thomas, France
Patrick D. Trevor-Roper, England
Jun Tsutsui, Japan
Alberto Urrets-Zavalia, Jr., Argentina
Jorge Valdeavellano, Peru
Antoon A. J. J. van der Eerden, Holland
Juan Verdaguer, Chile
Georges Vouvalis, Tunisia
J. D. Weintraub, Ohio
Darcy Wilson, New York
M. Osman Youssef, Egypt

SUB-COMMITTEE ON NOISE IN INDUSTRY OF THE COMMITTEE ON CONSERVATION OF HEARING
October 1, 1954, to March 31, 1955

RESEARCH

Those of you who read the last Quarterly Report will remember that I promised to make a report on the progress of the organization of the Sub-Committee's full-time staff. I would like to divide this report on progress into three main sections; namely, personnel, physical setup, and equipment.

Personnel

The accompanying organizational chart will show the way in which the personnel have been assigned. Recently we have made use of student help on an hourly basis. Many odd jobs which are not worthy of the time of the highly trained professional staff can be done by student help. For example, punching the IBM cards is a manual operation which requires little or no training and is certainly a waste of time for a highly trained statistician. At present, we are using a man from a local business school who spends three or four hours every afternoon punching IBM cards. Various odd jobs in the laboratory are done by a student from the audiology department of a local college. Through the use of this type of help, we can assure ourselves of assistance when we need it without committing ourselves to full-time personnel. I am happy to say this plan is working out nicely.

Biographical sketches outlining the background of the professional members of the staff are appended to this report.

There are no changes in the membership of the Sub-Committee itself except the addition of three liaison members. Mr. Floyd Frazier represents the insurance organizations who are contributing to the support of the research staff.

Mr. Frazier will also act as a coordinator between the Sub-Committee on Noise in Industry and any of the industries who deal with the insurance companies represented by Mr. Frazier. We are indeed fortunate to have an arrangement of this kind. It will save a great deal of correspondence and administrative work by the Research Staff of the Sub-Committee. If any of the companies concerned wish to contact the Sub-Committee, they may do so through Mr. Frazier; and when members of the Sub-Committee wish to contact any of the

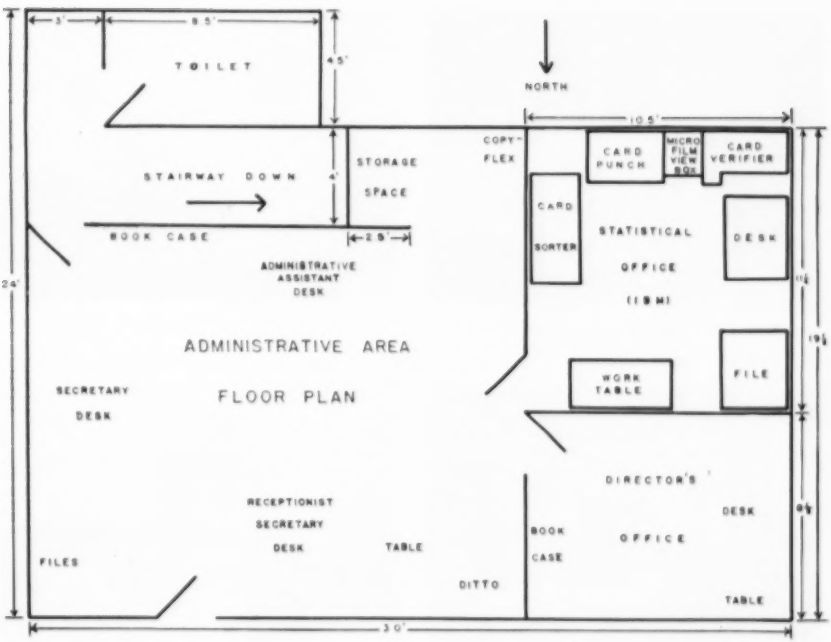


FIGURE 1.

firms represented, they may, in turn, do so through Mr. Frazier. This type of channeling establishes very efficient and effective cooperation.

In September 1954, during a meeting of the American Academy of Ophthalmology and Otolaryngology, Dr. Howard House and I were approached by Mr. Clark Bridges of the Council on Industrial Health of the American Medical Association regarding the formation of a committee in the American Medical Association to deal with the noise problem from the standpoint of the industrial physician. On our advice, Mr. Bridges returned to the Council on Industrial Medicine with the recommendation that in place of setting up a full committee, the American Medical Association appoint a member of the Council on Industrial Medicine to act as a liaison between the Sub-Committee on Noise in Industry and the Council on Industrial Medicine of the American Medical Association. Needless to say, the Sub-Committee was very happy with this arrangement, since it added considerable prestige to our group and prevented organizing another committee, which would seem to be redundant.

About a month ago Doctor House received a letter from Dr. William McNally of Montreal, Canada, suggesting a liaison type of connection between the Canadian Ear, Nose and Throat Society and the Sub-Committee on Noise in Industry. This appeared to be an excellent idea, and at Doctor McNally's suggestion, Dr. Harold McCoy of Montreal General Hospital was appointed as the Canadian liaison. We hope that Doctor McCoy will be able to be present at our

FIG. 2—Outside of building.

FIG. 3—Administrative office.

FIG. 4—Statistical office.

FIG. 5—Card punching; card verifying.

FIG. 6—IBM equipment: card punch; microfilm viewer; card verifier.

Sub-Committee meetings so that we may exchange information to our mutual benefit. As Director of the full-time staff of the Sub-Committee on Noise in Industry, I am very gratified by the addition of the three liaison members. They will add considerably to our information as well as to our prestige.

Physical Facilities

The next portion of the report will deal with the physical setup of the research headquarters. It has been said many times that a picture is worth a good many thousand words. I hope, therefore, that the diagram and pictures which will follow will do a great deal more than a verbal description. As you will see by figures 1 and 7, the space is set off in two parts. Figure 1 shows the administrative section. This space is on the ground floor and projects north from the laboratory area. It is divided into three sections. The large section houses the two secretaries and the ad-

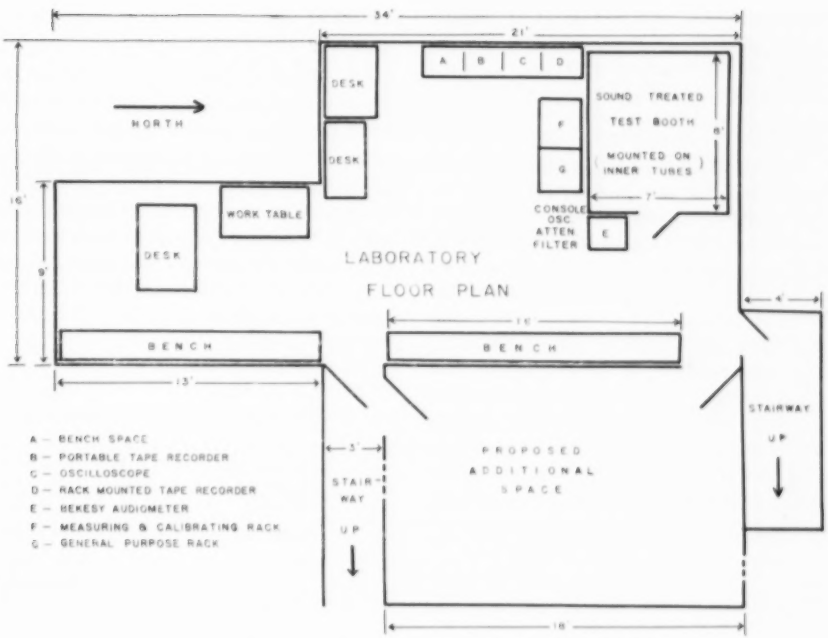


FIGURE 7.

ministrative assistant, with all the equipment pertinent to these jobs. The area marked statistical office and IBM, contains the IBM equipment, the data files and the working space for the statistician. The third area, marked Director's office, contains the usual furniture of a standard administrative office. The partitions are made of gypsum board, the ceiling and upper walls have been treated with acoustical tile. The floor is covered with rubber tile. Needless to say, we are very comfortable in our new quarters, which are very convenient to the laboratory. True, the IBM section is a little crowded and, perhaps, a little noisy when the sorter or the punching machines are being operated. However, these operations are not continuous and, therefore, they do not cause too much inconvenience (figs. 2, 3, 4, 5, 6).

Figure 7 pictures the laboratory space and proposed addition which will be used for study areas and a permanent measurements room. We expect this addition to supply us with approximately 250 square feet. All of the laboratory space is in a sub-basement which is one level below the street on the east side and two levels below the main traffic artery which is south of the building. You will notice a sound treated booth in one corner. Measurements taken in this sound treated booth show levels which are beneath those measurable by standard sound level meters. The entire laboratory, including the ceiling and walls, is lined with two-inch-thick fiberglass material. The floor is covered with cork tile and the solid wooden doors are extra heavy. There are no exits from the laboratory other than the two doors. The

laboratory space in general is ideally arranged for our type of research, because of its location and low ambient noise levels (fig. 8).

Facilities for Handling Data

During the past year many companies have been organizing hearing conservation programs, and consequently, there is considerable data in the field more or less acceptable for our purposes. After a survey of the field conditions and the type of data available has been completed, there still remains the problem of getting the data to our headquarters. We have made arrangements for microfilming any records which may be acceptable for our uses. The Recordak Company maintains facilities in most of the larger cities in the United States, and we can rent the microfilming equipment wherever the material happens to be, which makes it possible to bring large amounts of data to the laboratory in very small packages. Such a system lends itself very well to our needs, for it allows the company to retain the data in its own files and thus to avoid being without the records or losing them in transit. It also answers the problem of file space, since these data on standard cards would require a great deal of room, while the microfilm is easily stored in a very small space. After the data have been transported to the laboratory, the film is placed in a microfilm viewer, which enlarges the picture to a readable size, and the card punching is done directly from the enlarged pictures. In this manner we

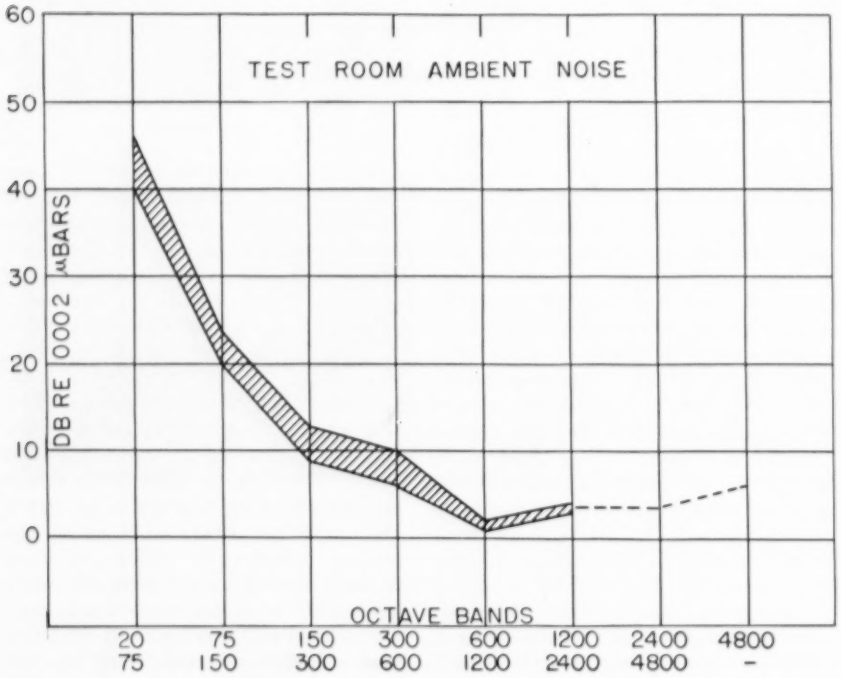


FIGURE 8.

are able to obtain a sizable amount of data (fig. 6).

We are also well equipped to duplicate any of our data or any of the materials coming from the laboratory for distribution or other uses. We have a standard Ditto machine and a recently acquired Copyflex, manufactured by the Bruning Company. This instrument is particularly useful when we have need of several copies for distribution to the Sub-Committee members or to the larger parent Committee on Conservation of Hearing.

At present, we have a card punch machine with numerical and alphabetical facilities combined with a duplicator, as well as a card verifier and a card sorter. This will allow us to handle large amounts of data easily. Those operations which require other equipment are rare and, if necessary, can be done by the IBM headquarters, which is close to our laboratory.

The following section will deal with equipment which has been recently acquired. Again, I shall depend upon pictures to demonstrate the arrangement of the equipment in its present form. Naturally, when the proposed addition is finished, some of the equipment will be housed in the measurements room, but at present it is arranged as seen in figures 9, 10 and 11.

During the past quarter we have obtained equipment which makes it possible to do complete noise analyses in the field and in the laboratory. This includes any type of noise from the steady state continuous to the intermittent impact noise. With the advice of our acoustics physicist, we have developed a standard operating procedure for recording and measuring any type of noise which we may be asked to study. We are also equipped to do complete studies on the attenuation of ear plugs or helmets. We can do audiometry in the laboratory or in the field, including both pure tone and speech via earphone or free field. All of our equipment can be calibrated, whether it deals with noise measurement or the measurement of hearing. We

FIG. 9—Laboratory equipment.

FIG. 10—Laboratory equipment and sound treated booth (door open).

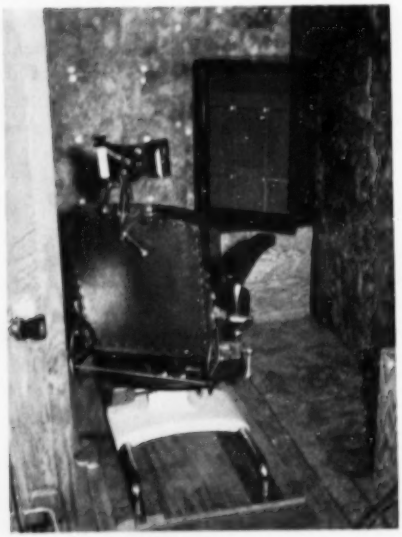


FIG. 11—Interior of sound treated booth.

probably have the finest specialized laboratory of its kind in the country. Within the next year or two, we hope that we will be able to demonstrate that this organization is well able to use its facilities and produce good data upon which to base conclusions regarding the noise problem.

Wisconsin State Fair Project

The data which we obtained on the 3600 cases seen at the Wisconsin State Fair are nearly all transferred to IBM cards. All but a few of the records are punched and verified, and we will soon be running correlations on these data. We have high hopes for these, and I feel they will be justified.

Educational Progress

The Quarterly Reports have received a very excellent response. The first Quarterly Report, which summarized the activities of the Sub-Committee prior to April 1, 1954, has been sent out to 500 individuals and organizations. The second report, dealing with the activities of the Sub-Committee staff from April 1, 1954, to July 1, 1954, has been circulated to 600 individuals and organizations. The last or Third Quarterly

Report has been mailed to over 800 individuals and organizations. It is gratifying to see that the information contained in these reports is apparently of interest and value to those individuals and organizations concerned with the noise problem.

The film, "Your Ear and Noise," which was made and produced by the Sub-Committee on Noise in Industry, has been shown sixteen times during the period from October 1, 1954, to January 1, 1955. The demand for this film has increased so rapidly that we have had to acquire more copies. At present, we have four copies which are continuously in use at one place or another.

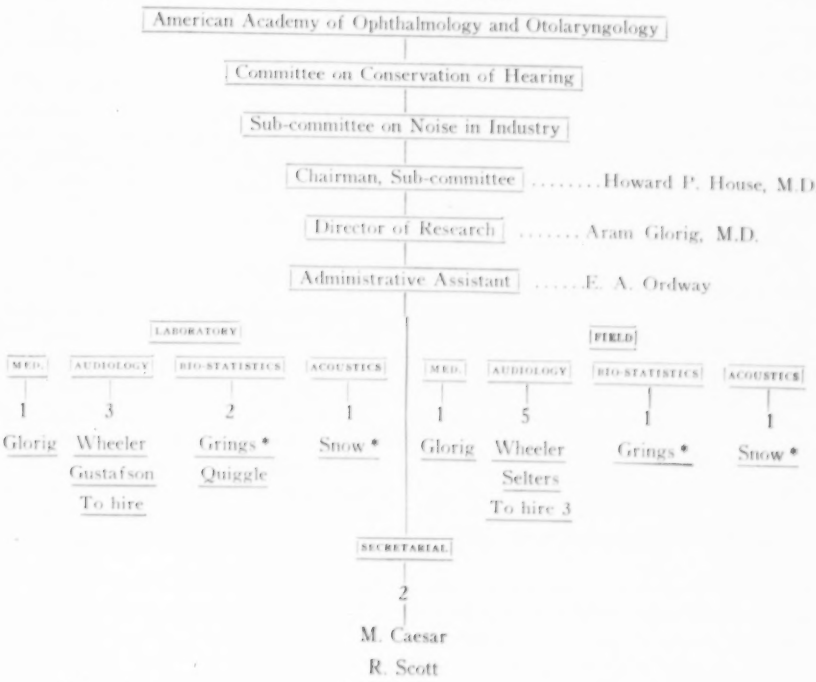
In closing, I would like to invite all of the readers of this report to send in suggestions regarding changes or additions. Next time I hope to be able to report on some of the findings of the Wisconsin State Fair Project.

ARAM GLORIG, M.D.
Director of Research

COMMITTEE AND STAFF

Howard P. House, M.D., Chairman
Professor of Otolaryngology
University of Southern California
Hallowell Davis, M.D.
Director of Research
Central Institute for the Deaf

TABLE OF ORGANIZATION



* Part time

Kenneth Mosier Day, M.D.
Professor of Otology
University of Pittsburgh
Meyer S. Fox, M.D.
Department of Otolaryngology
Marquette University
Stacy R. Guild, Ph.D.
Associate Professor of Otology
Johns Hopkins University
Gordon D. Hoople, M.D.
Professor of Otolaryngology
Syracuse University
Dean M. Lierle, M.D.
Professor of Otolaryngology
University of Iowa
C. Stewart Nash, M.D.
Department of Otolaryngology
University of Rochester
Walter A. Rosenblith
Massachusetts Institute of Technology

Office of the Chairman
1136 West Sixth Street
Los Angeles 17, California

Full-Time Research Staff Members
Aram Glorig, M.D.
Director
Douglas E. Wheeler, Ph.D.
Field Representative
William W. Grings, Ph.D.
Biostatistician
William B. Snow, B.S., E.E.
Physicist
John A. Gustafson, M.S.
Weldon Selters, M.A.
Audiologists
Robert Quiggle, M.A.
Statistician
Edgar A. Ordway
Administrative Assistant
Marjorie A. Caesar
Ruth A. Scott
Secretaries

Office of the Research Staff
11 North Bonnie Brae Street
Los Angeles 26, California

SECTION ON INSTRUCTION

SIXTIETH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 9-14, 1955
PALMER HOUSE
Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1955 *dues may order instruction tickets in advance of the meeting.* Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 7.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Orders will not be accepted for presale after October 4. To ensure acceptance they should be mailed by October 1 to William L. Benedict, M.D., 100 First Avenue Building, Rochester, Minnesota.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two or three different days, allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third preferences for each period in case first preference is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

Tickets will be on sale during the meeting at the Instruction Desk in the Registration Foyer.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

CHANGE IN INSTRUCTION COURSE PROGRAM AS LISTED IN THE MARCH-APRIL 1955 TRANSACTIONS

Course 241 on "Surgical Importance of the Orbital Fascia" by Dr. Orville E. Gordon has been cancelled.

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Adler	Physiology of Vision	3 PERIODS M-4, T-4, W-4
2	Allen et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
3	Ascher	Aqueous Veins in Glaucoma	2 PERIODS M-5, M-6
4	Ballintine & Johnson	Clinical Aqueous Tonography	2 PERIODS W-4, W-5
5	Bedell	Medical Ophthalmoscopy	3 PERIODS M-4, T-4, W-4
6	Berke	Surgery of Oblique Muscles	2 PERIODS M-5, M-6
7	Berliner	Slit Lamp Microscopy: Advanced	4 PERIODS M-4, T-4, W-4, Th-4
8	Birge et al	Vascular Disease: New Theories	2 PERIODS M-4, M-5
9	Boeder	Practical Optics	2 PERIODS M-4, M-5
10	Brown et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
11	Callahan et al	Plastic Surgery: Fundamental	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
12	Clark, G.	Retinal Detachment	3 PERIODS T-6, W-5, Th-5
13	Clark, W.	Surgery of Ocular Muscles	2 PERIODS M-5, M-6
14	Cooper	Accommodative Esotropia	2 PERIODS M-4, M-5
15 or 16	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
17	Costenbader et al	Comitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
18	DeVoe	Diseases of Cornea	2 PERIODS M-5, M-6
19	Fink	Vertical Muscle Problems	3 PERIODS M-6, T-6, W-6
20	Friedenwald & Becker	Studies on Aqueous Flow	2 PERIODS M-4, M-5
21	Gordon	Practical Perimetry	4 PERIODS M-4, T-4, W-4, Th-4
22	Guibor	Orthoptics: Office & Home Techniques	3 PERIODS T-6, W-6, Th-6
23	Henderson	Neuro-ophthalmology	3 PERIODS M-4, T-4, W-4
24	Hughes et al	Ophthalmic Plastic Surgery: Advanced	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
25	Knighton et al	Highlights in Glaucoma	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
26	Lyle	Neurologic Lesions	2 PERIODS M-5, M-6

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
27	McLean, Guyton Maumenee,	Cataract Surgery	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
28	Meyer et al	Glaucoma	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
29	Ogle	Essential Aspects of Binocular Vision	2 PERIODS T-5, T-6
30	O'Rourke	Cross Cylinders & Astigmatic Dials	2 PERIODS T-5, T-6
31	Paton	Corneal Surgery	3 PERIODS M-6, T-6, W-6
32 or 33	Pfeiffer & Jones	Roentgenography of the Eye & Orbit	2 PERIODS T-5, T-6 or W-5, W-6
34	Pino & Edwards	Problems of Bifocal & Aphakic Lenses	2 PERIODS T-5, T-6
35	Pischel & Wadsworth	.....SOLD OUT.....	
36	Regan, J., & Regan, C.	Refraction: Practical Aspects & Aids in Difficult Problems	6 PERIODS T-4, T-5, W-4 W-5, Th-4, Th-5
37	Rolett	Gonioscopy: Practical Application	2 PERIODS W-5, W-6
38	Romaine et al	Ocular Motility	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
39	Rucker	Neuro-ophthalmology	4 PERIODS M-6, T-6, W-6, Th-6
40	Sanders, Gibson & Becker	Vascular Diseases	3 PERIODS M-6, T-6, W-6
41 or 42	Schepens	Indirect Ophthalmoscopy: Technique	2 PERIODS T-5, T-6 or W-5, W-6
43	Schimek & Bousquet	Gonioscopy & Aqueous Outflow Determinations	2 PERIODS Th-4, Th-5
44	Sloane	School Vision Testing	2 PERIODS M-4, M-5
45	Sloane	Telescopic Lenses	2 PERIODS T-4, T-5
46	Pollen & Gettes	Prescription of Prisms; Drugs in Refraction	2 PERIODS W-4, W-5
47	Stow & Moss	Diagnostic & Therapeutic Office Procedures	2 PERIODS M-4, M-5
48	Tait	Heterophoria & Comitant Heterotropia	2 PERIODS M-5, M-6
49	Theobald et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
50	Thorpe et al	Slit Lamp Biomicroscopy	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
51	Thorpe & Gray	Surgery of Intraocular Foreign Bodies	2 PERIODS W-5, W-6
52	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-4, M-5, T-4, T-5 W-4, W-5
TV53	Televised Clinic	Disorders of Motility	3 PERIODS M-4, M-5, M-6
TV54	Televised Clinic	Cataract Extraction	3 PERIODS T-4, T-5, T-6
TV55	Televised Clinic	Tonography and Surgery of Glaucoma	3 PERIODS W-4, W-5, W-6
TV56	Televised Clinic	Selected Ocular Surgery	3 PERIODS Th-4, Th-5, Th-6

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
206	Atkinson	Anesthesia
208	Barkan	Congenital Glaucoma
210	Berens	Cyclodiathermy, Iridencleisis, Cyclodialysis
221	Cusick	Ptosis
232	Fralick	Surgical Anatomy of Operations for Glaucoma
233	Fread	Ideal Refraction Equipment
235	Gaines	Ophthalmological Examination from Neuro-ophthalmological Viewpoint
249	Jampolsky	Ocular Divergence: Management
253	Kennedy	Eye Changes in Diabetes
262	Lemoine	Vitreous Opacities
270	Lowrey	Tropical Ophthalmology
276	Martens	Problems in Presbyopia
281	Mullen	Ophthalmic Emergencies
291	Rizzuti	Tumors of the Lids and Ocular Adnexa
303	Tassman	Retinopathy in Diabetes
A	Home Study	Embryology, Anatomy and Neuroanatomy

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
205	Amenabar	Retinal Detachment: Localization & Closure of Tears
211	Bettman	Value of Goniotomies
212	Boyes	Problems in Eye Surgery
217	Chandler	Cataract Surgery
244	Harley	Lesions of 3rd, 4th & 6th Cranial Nerves
251	Joy	Cerebral Angiography in Ophthalmology
254	Kestenbaum	Reading Glasses
260	Leahey	Corneal Transplantation
265	Levy	Motion Picture Photography: Methods & Highlights
272	Macnie	Head Trauma with Ocular Symptoms
293	Rychener	The Lacrimal Apparatus
295	Simpson	Convergence Insufficiency & Divergence Excess
B	Home Study	Physiology

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Emotional Elements in the Ophthalmologist's Patients
204	Alvaro	Uveitis: Etiology & Treatment
209	Benton	Refraction: Handling the Difficult Patient
213	Calhoun	Dystrophies & Degenerations of the Cornea
219	Coston	Inflammatory & Degenerative Disease of Retina: Differential Diagnosis & Treatment
225	Davis	Sutures in Cataract Surgery: Types & Uses
226	Dixon	Indirect Ophthalmoscopy in Office Routine & in Intraocular Surgery
234	Fry	Lesions at Posterior Pole of the Eye
238	Gipner	Recent Advances in Ophthalmology
252	Katz	Recent Advances in Ophthalmic Anatomy: Clinical Application
255	Keyes	Retinal Vascular Disease: Ophthalmoscopic Studies
261	Leinfelder	Papilledema & Optic Neuritis
275	Mann, W.	Optical Correction of Aphakia
292	Rundles	Streak Retinoscopy
297	Solanes	Hypertension: Fundus Changes & Postmortem Findings
299	Spaeth	Plastic Surgery: Practical Aspects
301	Swan	Ophthalmic Therapeutics
310	Whalman	Minor Surgical Procedures of the Eye
C	Home Study	Refraction

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
207	Bannon	Aniseikonia
208	Barkan	Congenital Glaucoma
218	Cordes	Eye Changes in Diabetes
221	Cusick	Ptosis
229	Egan	Cross Cylinder: Application & Theory
230	Falls	Ocular Geriatrics
233	Freed	Ideal Refraction Equipment
243	Haik	Intraocular Foreign Bodies
247	Hogan	Lesions of the Choroid: Their Pathologic Significance
249	Jampolsky	Ocular Divergence: Management
253	Kennedy	Eye Changes in Diabetes
262	Lemoine	Vitreous Opacities
266	Lewis	External Diseases
276	Martens	Problems in Presbyopia
281	Mullen	Ophthalmic Emergencies
289	Post	Refraction: Difficult Problems
296	Simpson	Probing of Nasolacrimal Duct in the Adult
300	Steffensen	ACTH, Cortisone & Hydrocortisone
303	Tassman	Retinopathy in Diabetes
D	Home Study	External Diseases, Bacteriology & Virology

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
205	Amenabar	Retinal Detachment: Localization & Closure of Tears
211	Bettman	Value of Goniotomies
212	Boyes	Problems in Eye Surgery
222	Cutler	Cataract: Methods of Extraction
228	Dunnington	Cataract Extraction: Influences Upon End Results
244	Harley	Lesions of 3rd, 4th & 6th Cranial Nerves
250	Jardon	Fitting Ocular Prostheses
254	Kestenbaum	Reading Glasses
260	Leahey	Corneal Transplantation
268	Lloyd	Diseases of the Cornea
271	MacLean	Practical Aspects of Cataract Surgery
272	Macnie	Head Trauma with Ocular Symptoms
279	Merriam	Radiotherapy
283	Olson, J.	Iritis, Iridocyclitis, Keratitis
290	Randolph	Advanced Glaucoma: Treatment
293	Rychener	The Lacrimal Apparatus
302	Tabor	Vertical Muscles
309	Weston	Clinical Measurement of Astigmatism
E	Home Study	Perimetry and Neuro-ophthalmology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
204	Alvaro	Uveitis: Etiology & Treatment
213	Calhoun	Dystrophies & Degenerations of the Cornea
225	Davis	Sutures in Cataract Surgery: Types & Uses
226	Dixon	Indirect Ophthalmoscopy in Office Routine & in Intraocular Surgery
231	Fonda	Subnormal Vision: Examination Technique
238	Gipner	Recent Advances in Ophthalmology
240	Goar	Monocular Cataracts: Management
261	Leinfelder	Papilledema & Optic Neuritis
263	Leopold	Advances in Therapy
264	Lepard	Eye Diseases Common to Children
267	Linksz & Tait	Telescopic Lenses & Magnifying Systems
275	Mann, W.	Optical Correction of Aphakia
277	McCaslin	Ocular Anesthesia
278	McGraw	Contact Lenses
286	Paulson	Lighting for Comfortable & Accurate Seeing
292	Rundles	Streak Retinoscopy
298	Spaeth	Concomitant Strabismus
313	Emerson	Ophthalmic Optics
F	Home Study	Extraocular Muscles

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
203	Allen, H.	Operating Room Technique
214	Cassady	Dacryocystitis & Lacrimal Drainage
220	Cunningham	Types of Head Pain
224	Davidson	Surgical Instruments: Preparation & Care
227	Dunlap	Divergence Anomalies
229	Egan	Cross Cylinder: Application & Theory
236	Gemeroy	External Diseases of the Eye
239	Givner	Skin & Eye Manifestations of Systemic Disease
248	Jacobson	Electroretinography
256	Klien	Clinical Pathology of the Retina
258	Kronenberg	Practical Ocular Therapeutics
266	Lewis	External Diseases
274	Mann, M.	Hypertensive Vascular Changes in the Fundus
280	Morrison	Office Management of Glaucoma
304	Theodore	Ocular Therapeutics
305	Townes	Cataract Surgery: Complications
306	Uihlein	Neuralgias of the Head: Treatment
307	Veirs	External Dacryocystorhinostomy & Corrective Procedures for Epiphora
G	Home Study	Ophthalmoscopy & Medical Ophthalmology

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
215	Chamberlain	Vertical Motor Anomalies
216	Chamlin	Perimetry: Detection of Minimal Defects
222	Cutler	Cataract: Methods of Extraction
223	Daily	Biomicroscopy of Vitreous & Fundus Oculi
237	Gifford	Surgical Procedures on Lacrimal Apparatus
241	Gordon, O.	..... CANCELLED .....
242	Hague	Beta Irradiation
245	Harris	Anesthetic Agents & Techniques
246	Havener	Abnormalities of the Optic Disc
273	Mangold	Proper Fitting of Glasses
285	Paton	Corneal Transplants: End Results
308	Wendland	Chiasmal Lesions
312	Wood	Macular Lesions
H	Home Study	Pathology

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
202	Alfano	Internal Carotid Artery
257	Kornzweig	Practical Embryology
259	Kronfeld	Glaucoma Surgery: End Results
264	Lepard	Eye Diseases Common to Children
267	Linksz & Tait	Telescopic Lenses & Magnifying Systems
287	Perera	Retinal Detachment
288	Posner	Glaucomatocyclitic Crises
294	Scheie	Surgery for Congenital Glaucoma
298	Spaeth	Concomitant Strabismus
300	Steffensen	ACTH, Cortisone & Hydrocortisone
302	Tabor	Vertical Muscles
311	Wise	Fundus Changes of the Optic Nerve Head
I	Home Study	Pharmacology & Therapeutics

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
214	Cassady	Dacryocystitis & Lacrimal Drainage
224	Davidson	Surgical Instruments: Preparation & Care
227	Dunlap	Divergence Anomalies
236	Gemeroy	External Diseases of the Eye
256	Klien	Clinical Pathology of the Retina
258	Kronenberg	Practical Ocular Therapeutics
269	Lloyd	Fundus Lesions of the Senile Eye
274	Mann, M.	Hypertensive Vascular Changes in the Fundus
280	Morrison	Office Management of Glaucoma
283	Olson	Iritis, Iridocyclitis, Keratitis
287	Perera	Retinal Detachment
294	Scheie	Surgery for Congenital Glaucoma
305	Townes	Cataract Surgery: Complications
306	Uihlein	Neuralgias of the Head: Treatment
307	Veirs	External Dacryocystorhinostomy & Corrective Procedures for Epiphora
J	Home Study	Optics & Principles of Biomicroscopy

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
215	Chamberlain	Vertical Motor Anomalies
223	Daily	Biomicroscopy of Vitreous and Fundus Oculi
237	Gifford	Surgical Procedures on Lacrimal Apparatus
242	Hogue	Beta Irradiation
273	Mangold	Proper Fitting of Glasses
282	Myers	Vertical Phorias & Tropias
308	Wendland	Chiasmal Lesions
311	Wise	Fundus Changes of the Optic Nerve Head
312	Wood	Macular Lesions
J	Home Study	Continued

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Orders will not be accepted for presale after October 4. To ensure acceptance they should be mailed by October 1 to

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

Academy Candidates and Guests May Purchase Tickets Only at Instruction Desk in Registration Foyer

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 7

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday
- ☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in oto-laryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.

2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1955 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.

3. Make check payable to W. L. Benedict, M.D.

4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD		FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
		Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON.	4						
P.M.	5						
	6						
TUES.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
WED.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
THURS.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						

CONTINUOUS COURSES AND PANEL DISCUSSIONS
OTOLARYNGOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
101	Boies et al	Rhinologic Problems	2 PERIODS Th-2, Th-3
102 or 103	Cohen	Rhinoplasty Complications	2 PERIODS T-1, T-2 or W-1, W-2
104 or 105	Conley, Daly & Martin	Carcinoma of Head & Neck	2 PERIODS T-1, T-2 or W-1, W-2
106	Converse	Nasal Plastic Surgery—Basic	2 PERIODS W-2, W-3
107	Converse	Nasal Plastic Surgery—Advanced	2 PERIODS Th-2, Th-3
108	Cullen et al	Anesthetic Emergencies	2 PERIODS T-2, T-3
109 or 110	Hansel	Allergy	2 PERIODS W-1, W-2 or Th-1, Th-2
111 or 112	Hardy & Pauls	Clinical Audiometry for Young Children	2 PERIODS T-2, T-3 or W-2, W-3
113	Hilding	Physiology of Hearing: Relation to Hearing Loss and Its Therapy	2 PERIODS T-1, T-2
114 or 115	Huffman	Plastic Surgery of Head & Neck: General Problems	2 PERIODS W-2, W-3 or Th-2, Th-3
116 or 117	Jackson	Cancer of Larynx, Bronchi & Esophagus	3 PERIODS T-1, T-2, T-3 or Th-1, Th-2, Th-3
118	Kazanjian et al	Traumatic Injuries of Face	2 PERIODS T-2, T-3
119	Lindsay	Vertigo	2 PERIODS Th-2, Th-3
120	Maxwell	Diseases of Salivary Glands: Diagnosis & Treatment	2 PERIODS Th-1, Th-2
121 or 122	Meltzer et al	Your Otological Office Problems: Panel Discussion	2 PERIODS T-1, T-2 or W-1, W-2
123	Miller et al	ENT Cancer Can Be Treated in Your Community: Panel Discussion	2 PERIODS T-2, T-3
124 or 125	Moses	Speech & Voice Therapy	2 PERIODS W-2, W-3 or Th-2, Th-3
126	Mueller et al	What the Otologist Should Know About Hearing Aids	2 PERIODS T-1, T-2
127 or 128	Pressman	Cancer of Head and Neck	2 PERIODS W-1, W-2 or Th-1, Th-2
129	Scheldrup	Anatomy of Neck	2 PERIODS W-1, W-2
130 or 131	Seltzer	A Typical Rhinoplasty	2 PERIODS T-2, T-3 or Th-2, Th-3
132 or 133	Walsh	What Can Be Done for Deafness	2 PERIODS T-2, T-3 or Th-2, Th-3
TV134	Televised Clinic	Surgery of Deformities of the Head and Neck	3 PERIODS T-1, T-2, T-3
TV135	Televised Clinic	Surgery of Carcinoma of the Larynx	3 PERIODS W-1, W-2, W-3
TV136	Televised Clinic	Surgery of Tumors of the Nose and Sinuses	3 PERIODS Th-1, Th-2, Th-3

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	The Adenoid Operation
403	Anderson	Streamlined Allergy History for Otolaryngologists
404	Baker	Chronic Nonspecific Laryngitis
409	Boyden	Nasal Operation for Relief of Malignant Exophthalmos
410	Brandenburg	Understanding the Problem Patient
415	Cody	Lesions of Eustachian Tube: Management
416	Cohen, B.	Septum Correction & Rhinoplasty: A Combined Procedure
421	Davies	Development of Pharynx in Man as a Basis for Rational Surgery
425	DeWeese	The Problem of the Dizzy Patient
443	Hoople	Pitfalls in Office Audiometry
446	House	Atresia of Ear Canal: Congenital & Acquired
448	Ireland	Common Sense Treatment of Office Patient
452	Jordan, L.	Nasal Fractures
460	Kinney	Hearing Impairments in Children
462	Kos	Management of Chronic Otorrhea
467	Lempert	Temporal Bone Surgery
470	Lewy	Direct Laryngoscopy
476	McQuiston	Endaural Radical Mastoidectomy
488	Rawlins	Modern Concepts in ENT Allergy
493	Scheldrup	Autonomic Neuroanatomy of the Head & Neck
495	Schuknecht	Perceptive Deafness
504	Slaughter	Congenital Defects of Head & Neck
506	Tschiassny	Concepts & Tests of Labyrinthine Function

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
422	Davison	Ethmoid Sinus Disease: Treatment
438	Hilger	Direct Fixation of Facial Fractures
442	Holinger & Johnston	Endoscopic Aspects of Esophageal Disease
449	Johnson	Tonsillectomies for Adult Patients
453	Jordan, R.	Allergy in Otology
456	Keim	Management of Parotid Tumors
464	Lake	Chronic Suppurative Otitis Media: Office Treatment
473	Lowenthal	Lesser Facial Fractures
485	Peer	Common Facial Deformities: Surgical Management
491	Ryan	Headache
492	Schattner	Rhinoplasty by Reassembly of Nasal Elements
494	Schenck	Chronic Frontal Sinusitis
496	Sellers	The Dangerous Ear: What to Do About It
500	Simonton	End-Organ Deafness
505	Tremble	Practical Therapeutics in Office Practice
510	Wilson	Otoplasty for Protruding Ears

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
402	Alfaro	Office Design & Equipment; Techniques
405	Baylin	ENT Disorders: Use of Roentgenograms in Diagnosis
408	Bellucci	Submucous Resection of Nasal Septum
412	Bryan & Bryan	Diagnosis of Inflammatory Diseases of ENT by Intracellular Pathology
423	DeMere	Plastic Surgery Problems & Management in Everyday Practice
428	Furstenberg	Nasal Obstruction: Practical Considerations
433	Goodhill	Management of Tinnitus
440	Hill	Errors in Otolaryngology
441	Hilsinger	Vascular Headaches
455	Kasper	Dacryocystorhinostomy
458	Kelemen	Mechanical Injuries of Hearing Organ
466	LeJeune & Lynch	Technique of Intralaryngeal Surgery
472	Long	Antibacterial Agents in Otolaryngology
474	McLaurin	External Otitis
480	Nash	Otolaryngologic Mishaps
481	Neivert	Workshop for Deformed Instruments
483	Ogura	Laryngectomy & Neck Dissection
486	Perlman	Fluid in Middle Ear: Etiology & Management
490	Rinkel	Food Allergy in Otolaryngologic Practice
499	Simmons	Problem of the Bleeding Patient
507	Van Alyea	Embryology of ENT
512	Wynn	Facial Injuries: Primary & Secondary Plastic Repair

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	The Adenoid Operation
406	Becker	Plastic Surgery for Protruding Ears
410	Brandenburg	Understanding the Problem Patient
411	Brodnitz	The Actor & Singer in ENT Practice
413	Bryan & Bryan	Diagnosis of Neoplasms in ENT by Papanicolaou Smears
414	Canfield	Speech Audiometry for Practicing Otologist
415	Cody	Lesions of Eustachian Tube: Management
424	Devine	Resection of the Maxilla
426	Erich	Fractures of Facial Bones
439	Hilger	Therapeutic Approaches to the Inner Ear
454	Juers	Preservation of Hearing in Surgery for Chronic Ear Disease
462	Kos	Management of Chronic Otorrhea
467	Lempert	Temporal Bone Surgery
470	Lewy	Direct Laryngoscopy
476	McQuiston	Endaural Radical Mastoidectomy
454	Pearlman	ENT Complaints in Old Age
488	Rawlins	Modern Concepts in ENT Allergy
497	Seltzer	Making a Facial Mask
504	Slaughter	Congenital Defects of Head & Neck
506	Tschiassny	Concepts & Tests of Labyrinthine Function

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
419	Costen	Mandibular Joint Syndrome
422	Davison	Ethmoid Sinus Disease: Treatment
429	Gill	Mycotic Infections in Otolaryngology
437	Harbert	Clinical Audiology
446	House	Atresia of the Ear Canal: Congenital & Acquired
447	Hunnicutt	The Chronically Congested Nose
450	Jones, A.	Helpful Hints for Everyday Practice
451	Jones, L.	Tear Sac Surgery
456	Keim	Management of Parotid Tumors
463	Kremen	Cancer of the Tongue: Surgical Management
464	Lake	Chronic Suppurative Otitis Media: Office Treatment
473	Lowenthal	Lesser Facial Fractures
477	Miller	Laryngeal Emergencies in Infants & Children
489	Reger	Practical Office Audiometry
498	Shambaugh	Practical Office Routine in Otolaryngologic Problems
500	Simonton	End-Organ Deafness
501	Sisson	Carcinoma of the Head & Neck
503	Skolnik	Electrodiagnosis of the Facial Nerve
510	Wilson	Otoplasty for Protruding Ears

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
404	Baker	Chronic Nonspecific Laryngitis
405	Baylin	ENT Disorders: Use of Roentgenograms in Diagnosis
407	Beinfield	Nasal Hemorrhage: Management
418	Corgill	Laryngopharyngectomy & Bilateral Neck Dissection
423	DeMere	Plastic Surgery Problems & Management in Everyday Practice
428	Furstenberg	Nasal Obstruction: Practical Considerations
430	Glorig	The Otolaryngologist & Hearing Aid Selection
434	Goodhill	Endaural Ear Canal Surgery
436	Hampsey	Sinusitis in Children
444	Hosler	Cardiac Arrest
445	House	Surgery of the Chronically Discharging Ear
459	Kemler	Bilateral Thyrotomy for Carcinoma of Larynx
461	Kobrak	Treatment of Middle Ear Deafness
465	Lederer	Approaches to Diagnosis & Treatment in Prevention of Otolaryngologic Misadventures
468	Lempert	Factors Governing Successful Restoration of Practical Hearing by Surgical Intervention
475	McNally	Labyrinthine Tests
478	Moore	Anesthesia in Otolaryngology
479	Morrison	Laryngeal Paralysis
480	Nash	Otolaryngologic Mishaps
482	Neivert	Surgery of Bony Nasal Vault
491	Ryan	Headache
508	Van Alyea	Recent Advances in Management of Sinusitis
509	Von Leden	Mucoceles
512	Wynn	Facial Injuries: Primary & Secondary Plastic Repair

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
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411	Brodnitz	The Actor & Singer in ENT Practice
414	Canfield	Speech Audiometry for Practicing Otologist
417	Cohen, S.	Deviated Noses & Their Correction
424	Devine	Resection of the Maxilla
427	Fowler	Facial Paralysis & Tics
431	Glorig	The Otologist & the Noise Problem
435	Hallberg	Severe Nosebleed & Its Management
436	Hampsey	Sinusitis in Children
439	Hilger	Therapeutic Approaches to the Inner Ear
443	Hoople	Pitfalls in Office Audiometry
445	House	Surgery of the Chronically Discharging Ear
452	Jordan, L.	Nasal Fractures
454	Juers	Preservation of Hearing in Surgery for Chronic Ear Disease
457	Kelemen	Otitis Media: Pathologic Changes as Guide to Clinical Procedures
466	LeJeune & Lynch	Technique of Intralaryngeal Surgery
468	Lempert	Factors Governing Successful Restoration of Practical Hearing by Surgical Intervention
474	McLaurin	External Otitis
479	Morrison	Laryngeal Paralysis
490	Rinkel	Food Allergy in Otolaryngologic Practice
493	Scheldrup	Autonomic Neuroanatomy of the Head & Neck

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
419	Costen	Mandibular Joint Syndrome
441	Hilsinger	Vascular Headaches
442	Holinger & Johnston	Endoscopic Aspects of Esophageal Disease
448	Ireland	Common Sense Treatment of the Office Patient
450	Jones, A.	Helpful Hints for Everyday Practice
451	Jones, L.	Tear Sac Surgery
453	Jordan, R.	Allergy in Otology
460	Kinney	Hearing Impairments in Children
469	Levin	Speech & Voice: Management of Defects
471	Lillie	Sinusitis: Diagnosis & Office Treatment
477	Miller	Laryngeal Emergencies in Infants & Children
492	Schattner	Rhinoplasty by Reassembly of Nasal Elements
495	Schuknecht	Perceptive Deafness
498	Shambaugh	Practical Office Routine in Otolaryngologic Problems
502	Skolnik	Cleft Palate
511	Woodman	Glottic Obstruction

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
408	Bellucci	Submucous Resection of Nasal Septum
420	Crispell	Allergy in Children: Diagnosis & Treatment
426	Erich	Fractures of Facial Bones
432	Goldman	Vasomotor Rhinitis
434	Goodhill	Endaural Ear Canal Surgery
440	Hill	Errors in Otolaryngology
447	Hunnicutt	The Chronically Congested Nose
449	Johnson	Tonsillectomies for Adult Patients
459	Kemler	Bilateral Thyrotomy for Carcinoma of Larynx
461	Kobrak	Treatment of Middle Ear Deafness
465	Lederer	Approaches to Diagnosis & Treatment in Prevention of Otolaryngologic Misadventures
475	McNally	Labyrinthine Tests
478	Moore	Anesthesia in Otolaryngology
483	Ogura	Laryngectomy & Neck Dissection
489	Reger	Practical Office Audiometry
499	Simmons	Problem of the Bleeding Patient
505	Tremble	Practical Therapeutics in Office Practice

News - - - Notes

ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES

The 62nd Annual Convention of the Association of Military Surgeons of the United States will be held at Hotel Statler, Washington, D. C., November 7-9, 1955.

The Association is the only international society devoted to the military aspects of medicine, dentistry, nursing, veterinary medicine, and allied sciences. Noted speakers, appropriate ceremonies and a variety of entertainment will make this year's meeting attractive to attending members and guests. There will be an outstanding scientific program devoted to the latest advances and trends in the specialty of military medicine.

For the entertainment of the members and guests, a full schedule of events is being arranged. As in the past, one of the features is the Honors Night Dinner on November 9 at which the Sir Henry Wellcome Medal and Prize, the Gorgas Medal, the Stitt Award, the McLester Award, the Louis Livingston Seaman Prize, and the Founder's Medal will be presented.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

An Extraordinary General Assembly of the IOAT was held in Paris, May 9, 1955, at 28, Rue Saint-Dominique, under the chairmanship of Prof. G. B. Bietti of Parma, Italy.

Sir Stewart Duke-Elder, President of the International Council of Ophthalmology, and Prof. John C. Snyder of the Harvard University School of Public Health attended the sessions.

Tributes to M. A. F. Mac Callan, Honorary President, who died on March 31, 1955, were read and the Assembly observed a minute of silence in his honor.

Modification of the Statutes necessitated by the death of the President-Founder, by the obligations of growing internationalization, by the admission of the IOAT to the World Health Organization and by the increasing number of countries interested in the Organization's activities were adopted. They included the creation of three vice-presidencies, instead of one, and of the position of treasurer.

The officers composing the new council are:

President: G. B. Bietti, Italy
Vice-Presidents: Arnold Sorsby, Great Britain
Phillips Thygeson, United States of America
Roger Nataf, France

Secretary General: Jean Sedan, France

Associate Secretary: Yasushi Nakamura, Japan

Treasurer: Georges Farnarier, France

Headquarters of the IOAT will remain in London: 45, Lincoln's Inn Fields (W. C. 2).

All correspondence should be addressed to the Secretary General, 94, Rue Sylvabelle, Marseille, France.

The Assembly accepted as corresponding societies the following:

Societa Oftalmologica Italiana

Societad Oftalmologica Hispano-Americana

Philippine Ophthalmological Society

Seccao de Oftalmologia da Societade de Ciencias Medicas (Portugal)

Suomen Silmalaakariyhdistys (Finland)

New England Ophthalmological Society

The next Assembly will be in Madrid in 1956, during the Second Congress of the Societe Latine d'Ophtalmologie.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

The Sixth International Congress of Otolaryngology will take place in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957, under the presidency of Arthur W. Proetz M.D. The subscription for members is $25.00 (U. S. A.), which will include all official meetings of the Congress except the banquet. Relatives accompanying members may be registered as associates at a fee of $10.00.

The selected subjects for the Plenary (Combined) Sessions to be held Monday, Wednesday and Friday mornings will be Chronic Suppuration of the Temporal Bone, Collagen Disorders of the Respiratory Tract, and Papilloma of the Larynx. Outstanding internationally recognized authorities will open the discussion of each of these subjects.

Two types of communications are invited: (1) contributions to the discussions of the selected subjects, limited to 5 minutes; (2) original papers, limited to 15 minutes. These should be in one of the four official languages: English, French, German, Spanish.

Motion picture films will be shown each day except during the Plenary Sessions. There will be both scientific and technical exhibits.

An interesting program of tours, social functions and visits to points of interest in and around Washington has been arranged for those attending the Congress.

For further information please address the General Secretary, Paul H. Holinger, M.D., 700 N. Michigan Ave., Chicago 11, Ill.

CALIFORNIA

Dorhmann K. Pischel, M.D., has been appointed Head of the Division of Ophthalmology at Stanford University Medical School. Doctor Pischel received his B. A. degree from the University of California in 1918 and his M.D. from Stanford University Medical School in 1923. He has been associated with Stanford University Medical School since 1928.

KENTUCKY

The University of Louisville conferred an Honorary Doctor of Science Degree upon Dr. Frederic H. Verhoeff, Consulting Chief of Ophthalmology at the Massachusetts Eye and Ear Infirmary and Professor Emeritus of Ophthalmic Research at the Harvard Medical School, on the occasion of its commencement exercises, June 5, 1955.

LOUISIANA

The Mid-Winter Convention of the New Orleans Academy of Ophthalmology will be held January 3-6, 1956. "Diseases and Surgery of the Lens" will be the general topic to be discussed by Everett Kinsey, Ph.D., Detroit, Michigan; Frederick C. Cordes, M.D., San Francisco, California; Derrick Vail, M.D., Chicago, Illinois; John Dunnington, M.D., New York, New York; S. Rodman Irvine, M.D., Los Angeles, California; Harvey Thorpe, M.D., Pittsburgh, Pennsylvania; and Paul Chandler, M.D., Boston, Massachusetts. The program will include round table discussion periods.

The registration fee of $75 includes associate membership in the New Orleans Academy for the year 1956, as well as all other features of the convention. Advance registration should be secured by writing now to the New Orleans Academy of Ophthalmology, 211 South Saratoga St., New Orleans, Louisiana.

Hotel reservations should be made early by writing to Roosevelt Hotel, 123 Baronne Street, New Orleans.

MAINE

Colby College, Waterville, Maine, will present the Third Annual Course in Industrial Deafness, August 7 through 13, 1955. The objective of the course will be to train personnel in initiating and in conducting conservation hearing programs in noisy industries. Director of the course will be Frederick Thayer Hill, M.D., and Assistant Director will be Joseph Sataloff, M.D. Seven full-time instructors have been selected from authorities

in this field. The class will be limited to twenty participants.

Registrants will live on the college campus. The tuition fee of $200.00 includes payment for board and room. Applications should be made to Mr. William A. Macomber, Director, Division of Adult Education and Extension, Colby College, Waterville, Maine.

MINNESOTA

Dr. William L. Benedict, Executive Secretary-Treasurer of the American Academy of Ophthalmology and Otolaryngology, was honored by his Alma Mater, the University of Michigan, when the degree of Doctor of Laws was conferred upon him during the University Commencement, at Ann Arbor, Michigan, June 11, 1955.

PENNSYLVANIA

The Dean of the Graduate School of Medicine of the University of Pennsylvania announces the appointment of Dr. Edmund B. Spaeth, Professor of Ophthalmology and Chairman of The Department of Ophthalmology, as Professor Emeritus of Ophthalmology.

The Dean also announces the appointment of Dr. Irving H. Leopold as Professor of Ophthalmology and Chairman of the Department of Ophthalmology of The Graduate School, succeeding Doctor Spaeth.

VIRGINIA

The Virginia Society of Ophthalmology and Otolaryngology will meet November 29 through December 2, 1955, at the Medical College of Virginia, Richmond, Virginia. The secretary is Peter N. Pastore, M.D., 1200 East Broad Street, Richmond 19, Virginia.

The Gill Memorial Eye, Ear, Nose and Throat Hospital, Roanoke, Virginia, has completed its 28th Annual Spring Congress in Ophthalmology and Otolaryngology. There was a total attendance of 350 doctors and their wives from all over the United States and Canada. The next Congress will be held April 2 through 7, 1956.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

EXAMINING AND CERTIFYING BODIES

American Board of Ophthalmology

Chairman: Dr. Francis Heed Adler
Secretary: Dr. Merrill J. King, 56 Ivie Road, Cape Cottage, Maine
Practical Examination: October 4-8, 1955, Chicago
Written Examination: January, 1956

American Board of Otolaryngology

President: Dr. LeRoy Schall
Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City, Iowa
Oral Examination: October 3-7, 1955, Chicago

American Board of Plastic Surgery

Chairman: Dr. James T. Mills
Secretary-Treasurer: Dr. Bradford Cannon, 330 Dartmouth Street, Boston 16, Mass.
Corresponding Secretary: Estelle E. Hillerich, 4647 Pershing Ave., St. Louis 8, Mo.
General Examination: September 19-24, 1955, Philadelphia

Brazilian Board of Ophthalmology

Chairmen: Prof. Cyro de Rezende
Prof. Cyro de Rezende
General Secretaries: Dr. Rubens Belfort Mattos, Caixa Postal 4086, Sao Paulo, Brazil
Dr. Sylvio de Almeida Toledo, Braulio Gomes 25, Sao Paulo, Brazil

The Royal College of Physicians and Surgeons of Canada

President: Dr. John Hepburn
Honorary Secretary: Dr. James H. Graham, 150 Metcalfe Street, Ottawa 4, Ontario, Canada

Written Examinations

Monday and Tuesday, October 3rd and 4th. These examinations will be held at eleven centers in Canada: St. John's, Newfoundland, Halifax, Quebec City, Montreal, Kingston, Toronto, London, Winnipeg, Edmonton, Saskatoon and Vancounver.

Oral and Clinical Examinations

The oral and clinical portion of the examination will be held in Winnipeg the week commencing Monday, November 7th, and in Toronto the week commencing Monday, November 21st.

INTERNATIONAL

International Association of Eye, Ear, Nose and Throat Society Secretaries

President: Dr. Roderick Macdonald
Secretary-Treasurer: Dr. Daniel S. DeStio, 1006 Highland Building, Pittsburgh 6, Pa.
Time and Place: October 11, 1955, Palmer House, Chicago, Ill.

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris VIIe, France
Secretary-General: Prof. A. Franceschetti, Clinique Ophthalmologique de l'Université, Geneva, Switzerland

International Broncho-Esophagological Society

President: Dr. Antonio da Costa Quinta
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Time and Place: November, 1955, Buenos Aires

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. Francis L. Lederer
Secretary: Dr. Louis Savitt, 3403 Lawrence Avenue, Chicago 25, Ill.

International Council of Ophthalmology

President: Sir Stewart Duke-Elder
Secretary: Dr. Edward Hartmann, 2 Avenue Ingres, Paris (16e), France
Time and Place: The Council meets at the time and place of each International Congress (usually every 4 years) and other years alternately in France and Great Britain. (May 7, 1955, Paris, France)

International Federation of Ophthalmolgical Societies

President: Sir Stewart Duke-Elder
Secretary: Prof. Dr. Edward Hartmann, 2 Avenue Ingres, Paris (16e), France
Time and Place: September, 1958, Brussels, Belgium

International Organization Against Trachoma

President: Dr. A. F. MacCallan
Secretary-General: Professor Arnold Sorsby, 45 Lincoln's Inn Fields, London, W. C. 2, England

Pan-American Association of Ophthalmology

President: Dr. M. E. Alvaro
Acting Secretary-Treasurer: Dr. Daniel Snydacker, 109 North Wabash Avenue, Chicago 2, Ill.
Time and Place: January 9-14, 1956. Santiago, Chile

Pan-American Association of Ophthalmology, Puerto Rico Chapter

President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

PAN-AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY
President: Dr. Juan M. Tato
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pa.
Time and Place: Fifth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, San Juan, Puerto Rico. Spring, 1956. President of Congress, Dr. Juan Font, Puerto Rico

FOREIGN

THE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY OF ALBERTA
President: Dr. H. E. Dowling
Secretary: Dr. M. R. Marshall, North Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: April 1956, Calgary, Alberta, Canada

ALL-INDIA OPHTHALMOLOGICAL SOCIETY
President: Dr. Alice B. Laker
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, 21, Queen's Road, Bombay 4, India

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER
President: Dr. Leoncio de Souza ueiroz
First Secretary: Dr. Alberto Galo
Second Secretary: Dr. Aloisio Af. Ferreira

AUSTRIAN OTOLARYNGOLOGICAL SOCIETY, VIENNA
President: Prof. Dr. E. Schlander
Secretary: Dr. Julius Popper, Schottenring 17, Wien I., Austria
Time and Place: First Tuesday of the month. Vienna General Hospital, Alserstrasse 4, Vienna IX.

BOMBAY OPHTHALMOLOGISTS' ASSOCIATION
President: Dr. D. S. Sardesai
Secretaries: Dr. B. D. Telang and Dr. Y. K. C. Pandit, 395, Lamington Road, Bombay 4, India
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS
President: Mr. V. E. Negus
Secretary: Mr. Myles L. Formby, 45 Lincoln's Inn Fields, London W. C. 2, England

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Mr. J. H. Doggart
Secretary: Mr. B. Gluck, 76 Cathedral Road, Cardiff, Wales

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
Secretary: Dr. J. Clement McCulloch, 830 Medical Arts Building, Toronto, Ont., Canada

CANADIAN OPHTHALMOLOGICAL SOCIETY
President: Dr. P. B. Macfarlane
Secretary: Dr. R. G. C. Kelly, 113 St. Clair Avenue W., Toronto 5, Ont., Canada

CANADIAN OTOLARYNGOLOGICAL SOCIETY
President: Dr. G. Edward Tremble
Secretary: Dr. G. Arnold Henry, 170 St. George Street, Toronto, Ont., Canada

CENTRO DE ESTUDOS DE OFTALMOLOGIE DE SAO PAULO
President: Dr. Francisco Amendola
Secretary: Dr. Rubens Belfort Mattos, Caixa Postal 4086, Sao Paulo, Brazil
Time and Place: Every Tuesday evening at the Rua Condessa, Sao Joaquim 288, Sao Paulo

CHENGTU OPHTHALMOLOGICAL SOCIETY
President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan

CHINESE OPHTHALMOLOGY SOCIETY
President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEKING
President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT HEIDELBERG
President: Prof. Dr. med. W. Löhlein
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik
Time and Place: Generally in September in Heidelberg

FACULTY OF OPHTHALMOLOGISTS
President: Dr. John Marshall
Secretary: Mr. E. F. King, 45 Lincoln's Inn Fields, London, W.C. 2, England

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY
President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY
President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

IRISH OPHTHALMOLOGICAL SOCIETY
Secretary: Dr. L. B. Somerville-Large, 2 Fitzwilliam Place, Dublin, Ireland

ISRAEL OPHTHALMLOGICAL SOCIETY
President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. F. A. Williamson-Noble
Secretaries: Dr. R. D. Weeden Butler, and Dr. M. J. Roper-Hall, c/o Birmingham & Midland Eye Hospital, Church St., Birmingham 3, England
Place: Birmingham & Midland Eye Hospital, Church St., Birmingham 3, England

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. A. L. Guertin
Secretary: Dr. William Turnbull, 1414 Drummond St., Montreal, Que., Canada

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP
President: Prof. Dr. A. Hagedoorn
Secretary: Dr. P. G. Binkhorst, Sweelincklaan 20, Bilthoven, The Netherlands

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Mr. A. McKie Reid
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle-on-Tyne, Liverpool, Sheffield and Bradford

NORSKE OYELEGERS FORENING (SOCIETY OF NORWEGIAN OPHTHALMOLOGISTS)
President: Dr. Ludvig Wirsching
Secretary: Dr. Olav Lunde, Teatergaten 11, Oslo, Norway

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. C. Cormier
Secretary: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Three times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA
President: Miss Ida Mann
Secretary: Mr. Peter A. Rogers, 235 Macquarie Street, Sydney, New South Wales, Australia
Time and Place: October 17 to 21, 1955, Camberra, A.C.T., Australia

OPHTHALMOLOGICAL SOCIETY IN COPENHAGEN, DENMARK
President: Dr. G. Osterberg
Secretary: Dr. E. Vesterdal, Bülowsvej 18.B., Copenhagen, Denmark

OPHTHALMOLOGICAL SOCIETY OF EGYPT
President: Dr. Ismail Disouki
Honorary Secretary: Dr. Sabri Kamel, Dar El Hekmah, 42 Ksar El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ
Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, Mexico, D. F.
Time and Place: Second Friday of each month. Hospital de Nuestra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND
President: Dr. W. E. Carswell
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA
President: Dr. J. K. deKock
Secretary: Dr. J. Guillaume Louw, 17 Church Square, Cape Town

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM
President: Mr. O. Gayer Morgan
Secretaries: Derek Ainslie, M.D., and Charles A. Cook, 45 Lincoln's Inn Fields, London, W. C. 2

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN
President: Dr. Karl Safar
Secretary: Dr. F. Schwab, Univ. Augenklinik, Alsterstrasse 4, Vienna 9, Austria
Time and Place: Vienna, Austria, Alserstrasse 4. Second Monday of each month

OSTERREICHISCHEN OPHTHALMOLOGISCHEN GESELLSCHAFT
President: Prof. Dr. A. Pillat
Secretary: Dr. Frank Schwab, 1. Univ. Augenklinik, Wien

OXFORD OPHTHALMOLOGICAL CONGRESS
Master: Mr. Frank W. Law
Honorary Secretary and Treasurer: Mr. Ian C. Fraser, 21 Dogpole, Shrewsbury, Shropshire

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Carlos V. Yambao
Secretary-Treasurer: D. Jesus V. Tamesis, North General Hospital, Manila
Time and Place: December, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY
Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

PUERTO RICAN OPHTHALMOLOGICAL SOCIETY
President: Dr. Roberto Buxeda
Secretary-Treasurer: Dr. Guillermo Pico, Ave. Ponce de Leon 654, Santurce, Puerto Rico

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Jose Pico
Secretary for Ophthalmology: Dr. Guillermo Pico, Ave. Ponce de Leon 654, Santurce, Puerto Rico
Secretary for Otorhinolaryngology: Dr. Antonio Rullan
Time and Place: 8:00 P.M. on second Wednesday of even months at the Puerto Rico Medical Association Building, Santurce, P. R.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY
President: Mr. Humphrey Neame
Secretary: Mr. R. T. Hewitt, 1, Wimpole Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY
President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB
President: Dr. C. W. Graham
Secretary: Dr. John D. Fraser, 31 Kingsborough Gardens, Glasgow, Scotland
Time and Place: Last Saturday of March, Edinburgh and last Saturday in October, Glasgow

SOCIEDAD ARGENTINA DE OFTALMOLOGIA
President: Dr. Eduardo J. J. Lagos
Secretary: Dr. Alberto C. Cremona, Santa Fe 1171, Buenos Aires, Argentina
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA
President: Dr. Alberto Gormaz B.
Secretary: Dr. Carlos Charlin V., Mc. Iver No. 175, Santiago, Chile

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORHINOLARINGOLOGIA
President: Dr. Alfonso Gaitan N.
Secretary: Dr. Félix E. Lozano, Apartado Aéreo No. 5601, Bogota, Columbia
Time and Place: Second Tuesday of each month, Auditorium, Clinica Oftalmologica Hospital del Salvador, Santiago

SOCIEDAD CUBANA DE OFTALMOLOGIA
President: Dr. Rafael Labrador Fuiz
Secretary: Dr. Guillermo De La Torre, Calle G. No. 506, Vedado, Havana, Cuba
Time and Place: First Thursday bi-monthly. Malecon 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA
President: Dr. Manuel de Rivas Cherif
Secretary: Dr. Emilio Remolina Lopaz, Apartado Postal No. 8112, Mexico, D. F.
Time: 8:30 p.m., first Tuesday of each month

SOCIEDAD NICARAGUIENSE DE OFTALMOLOGIA
President: Dr. Ricardo Lacayo
Secretary: Dr. Gilberto Cuadra, Clinica Especializada, Managua, D. N., Nicaragua, C. A.

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA
Chairman: Dr. Alberto Urrets-Zavalia, Sr.
Secretary. Dr. R. Obregon Oliva, 9 de Julio 560, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA
President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jalisco, Mexico
Place: Edificio Lutencia Despacho 101

SOCIEDAD OFTALMOLOGICA HISPANO-AMERICANA
President: Dr. Manuel Lopez Enriquez
Secretary: Dr. Carlos Costi Garcia de Tunon, Montalban, 7, Madrid, Spain

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL
President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDAD OFTALMOLOGICA DE VALPARAISO
President: Prof. Dr. Jean H. Thierry
Secretary-Treasurer: Dr. Jorge Oyarzun O, Hospital de Ojos, Calle Blas, Cuevas, Valparaiso

SOCIEDAD DE OFTALMOLOGIA Y OTORRHINOLARINGOLOGIA DEL PARAGUAY
President: Dr. Crispin Insaurralde
Secretary: Dr. Honorio Campuzano, Coronel Bogado 492, Asuncion, Paraguay

SOCIEDAD PERUANA DE OTO-RINO-LARINGOLOGIA Y OFTALMOLOGIA
President: Dr. Enrique Cipriani V., Pasaje Olaya 156, Lima, Peru
Secretario General: Dr. Enrique Haro
Secretario de Actas: Dr. Alberto Castillo R.

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA
President: Dr. Paulo C. Pimental
First Secretary: Dr. Pedro Moacyr de Aguiar, Ladera Santa Thereza 136, Apt. 101, Rio de Janeiro
Second Secretary: Dr. Luiz Enrico Ferreira
Time: Third Friday of every month from April to December

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCHO-ESOFAGOLOGIA
President: Dr. Alberto Luis de Mendonca
Secretaries: Dr. Antonio da Costa Quinta and Dr. Albano Coelho, Avenida da Liberdade, 65, Lisbon 1, Portugal

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS
President: Dr. Amelio Bonfioli
Secretary: Dr. Mariana Noronha, Pauso Alegre 1150, Belo Horizonte, Brazil Minas Gerais
Place: Belo Horizonte, Minas Gerais

SOCIEDADE DE OFTALMOLOGIA DEL NORTE
President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA
President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARINGOLOGIA DO RIO GRANDE DO SUL
President: Dr. Mario Araujo Azambuja
First Secretary: Dr. Humberto Lubisco, Rua Donna Laura 45, Porto Alegre, Est., Rio Grande do Sul
Second Secretary: Dr. Ivo Kuhl

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO
President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paula Aranha de Azevedo, Xavier de Toledo 71,1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OTO-RHINO-LARINGOLOGIA DE RIO DE JANIERO
President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIETA OFTALMOLOGICA ITALIANA
President: Prof. Giuseppe Ovia
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE
President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium

SOCIETE FRANCAISE D'OPHTALMOLOGIE
Secretaire-général: Dr. Marcel Kalt, 81 rue Saint Lazare, Paris (9) France

SOCIETE FRANCAISE D'OTO-RHINO-LARYNGOLOGIE
President: Dr. Louis Leroux
Secretary-General: Dr. H. Flurin, 19 Ave. MacMahon XVIIe, Paris, France
Place: 17 rue de Buci, Paris, France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS
President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm So, Sweden
Time: Annual meeting, beginning of June

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND
President: Mr. W. E. Heath
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, Hants, England

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY
Chairman: Dr. Morris Shusterman
Secretary: Dr. George Thompson, Zoo St. Clair Ave., West Toronto, Ont., Canada

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Algernon B. Reese
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 9-14, 1955, Palmer House, Chicago, Ill.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION
President: Dr. Daniel S. Cunning
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia 3, Pa.
Time and Place: Annual meeting, May 15 and 16, 1956, Sheraton Mount Royal Hotel, Montreal, Que., Canada

AMERICAN LARYNGOLOGICAL ASSOCIATION
President: Dr. Bernard J. McMahon
Secretary: Dr. Harry P. Schenck, 326 S. 19th Street, Philadelphia 3, Pa.
Time and Place: May 13-14, 1956, Seigniory Club, Province of Quebec, Canada

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.
President: Dean M. Lierle
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.
Time and Place: May 15-17, 1956, Montreal, Canada

Eastern Section
Chairman: Dr. John A. Murtagh
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia 3, Pa.
Time and Place: January 13, 1956, Statler Hotel, Boston, Mass.

Middle Section
Vice-President: Dr. William H. Craddock
Secretary: Dr. Charles E. Kinney, 10515 Carnegie Avenue, Cleveland, Ohio
Time and Place: January 16, 1956, Netherland Plaza Hotel, Cincinnati, Ohio

Southern Section

Vice-President: Dr. J. M. Robison
Secretary: Dr. James A. Harrill, Bowman Gray School of Medicine, Winston-Salem, N. C.
Time and Place: The Shamrock Hotel, Houston, Texas

Western Section

Vice-President: Dr. David D. DeWeese
Secretary: Dr. Francis Sooy, 490 Post Street, San Francisco, Calif.
Time and Place: County Medical Society Bldg., San Francisco, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Erling Hansen
Secretary: Dr. Harold G. Scheie, 313 S. 17th St., Philadelphia 3, Pa.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Francis W. Davison
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: Atlantic City, N. J.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Alan C. Woods
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th Street, New York 19, N. Y.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. William J. McNally
Secretary-Treasurer: Dr. Lawrence R. Boies, 90 South Ninth St., Minneapolis 2, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Time and Place: May 11 and 12, 1956, the Seigniory Club, Province of Quebec, Montreal, Canada

AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY

President: Dr. Joseph Gilbert
Secretary: Dr. Louis Joel Feit, 66 Park Avenue, New York 16, N. Y.
Place: New York City

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. F. Lambert McGannon
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo, N. Y.
Time and Place: October 14, 1955. Palmer House, Chicago, Ill.

NATIONAL COMMITTEE FOR RESEARCH IN EYE DISEASES AND DISABILITIES

Chairman: Dr. James H. Allen
Secretary: Dr. L. V. Johnson, University Hospitals, Cleveland, Ohio

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. William F. Hughes
Secretary: Dr. Lorand V. Johnson, 10515 Carnegie Ave., Cleveland 6, Ohio
Time and Place: June, Atlantic City, N. J.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS INC.

President: Mr. Mason H. Bigelow
Executive Director: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.
Time and Place: March 26-28, 1956, Palmer House, Chicago, Illinois

SOCIETY OF MILITARY OPHTHALMOLOGISTS

President: Col. J. H. King, Jr.
Secretary-Treasurer: Capt. James A. Stokes, Walter Reed Army Hospital, Washington 12, D. C.
Time and Place: October 11, 1955. Palmer House, Chicago, Ill.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY

President: Dr. J. L. Custer
Secretary: Dr. W. W. Hart, Medical Arts Building, Shreveport, Ill.
Time and Place: First Monday of month, except January, June, July, September, at Confederate Memorial Hospital

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Albert K. T. Ho
Secretary: Dr. Robert H. Lee, 1413 Nuuanu Avenue, Honolulu
Time and Place: Third Thursday of each month, September through May (dinner meetings) 6:30 p.m., Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Ralph W. Pugmire
Secretary: Dr. Orson W. White, 1010 Medical Arts Building, Salt Lake City, Utah
Time and Place: Third Monday of each month, University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. E. Garland Walls
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.
Time and Place: April 13 and 14, 1956, Edgewater Gulf Hotel, Edgewater Park, Miss.

NASSAU OPHTHALMOLOGICAL SOCIETY

President: Dr. Frank Green
Secretary: Dr. Eugene T. Buckley, 131 Fulton Avenue, Hempstead, N. Y.
Time and Place: Every second month at various places

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Merrill J. King
Secretary: Dr. Karl Riemer, 403 Commonwealth Avenue, Boston, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Charles I. Johnson
Secretary: Dr. Burton E. Lovesey, 15 West Central Street, Natick, Mass.
Time and Place: Three times a year, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, Mass.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Lester T. Jones
Secretary: Dr. John F. Tolan, 3419 47th Ave. N.E., Seattle 5, Wash.
Time and Place: April 15-19, 1956, Phoenix, Ariz.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. J. Cortopassi
Secretary: Dr. Arno Weiss, 308 Eddy Bldg., Saginaw, Michigan
Time and Place: 6:30 p.m. every second Tuesday of each month, except summer months. Bancroft Hotel Saginaw

SIOUX VALLEY MEDICAL SOCIETY
President: Dr. T. H. Willcockson
Secretary-Treasurer: Dr. Sidney F. Becker, 603 National Bank Bldg., Sioux Falls, S. D.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Lyle M. Sellers
Secretary: Dr. G. S. Fitz-Hugh, 104 Market Street, Charlottesville, Va.
Time and Place: November, 1955, Houston, Texas

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. George G. Hankins
Secretary: Dr. J. G. Sellers, 413 Medical Arts Building, Norfolk, Va.
Time and Place: Quarterly. Norfolk Yacht and Country Club

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Otis M. Wilson
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.
Time and Place: September 10 and 11, 1955, Eagle River Resort, Eagle River, Wisconsin

STATE

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. David McCoy
Secretary: Dr. Frank S. Moody, 501 Medical Arts Building, Birmingham, Ala.

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. K. W. Cosgrove
Secretary: Dr. Max F. McAllister, 18 E. Dickson, Fayetteville, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. Robert N. Shaffer
Secretary: Dr. Channing Hale, 174 Nehama, Pomona, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. Katherine Chapman
Secretary: Dr. James C. Strong, Jr., 227 Sixteenth St., Denver, Colo.
Time and Place: Third Saturday of each month, September to May, at Colorado Medical Center, Denver

COLORADO OTOLARYNGOLOGICAL SOCIETY
President: Dr. D. F. Page
Secretary: Dr. Sidney H. Fieman, 1612 Tremont Pl., Denver, Colorado
Time: First Saturday of each month

CONNECTICUT STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. Sherburne Campbell
Secretary-Treasurer: Dr. Max Alpert, Professional Building, 881 Lafayette Street, Bridgeport, Conn.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Charles W. Boyd
Secretary: Dr. Kenneth S. Whitmer, 550 Brickwell, Miami, Fla.
Time and Place: Annual meeting, April 1956, Miami Beach, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Alton V. Hallum
Secretary: Dr. W. P. Rhyme, 403 Broad Avenue, Albany, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. P. Clark
Secretary: Dr. John J. Flick, 1443 N. Pennsylvania Street, Indianapolis, Ind.
Time: Annually in May

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. A. Thorson
Secretary: Dr. William C. Huffman, University Hospitals, Iowa City, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George F. Gsell
Secretary: Dr. David P. Trimble, 2nd Floor, Gazette Bldg., Emporia, Kan.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edwin B. Dunphy
Secretary: Dr. John E. McKeigue, 403 Commonwealth Avenue, Boston 15, Mass.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. R. Wallace Teed
Secretary: Dr. Anthony D. Calomeni, 309 Seymour Avenue, Lansing, Mich.
Time and Place: Three times a year in different cities

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Frank Adair
Secretary: Dr. Karl Sandt, 1750 Medical Arts Building, Minneapolis, Minn.
Time and Place: Second Friday of each month. Alternating Minneapolis and St. Paul

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. John J. Hammerel
Secretary: Dr. James D. Morrison, 28th Street & Poly Boulevard, Billings, Mont.
Time and Place: Annual meeting September 15 to 17, 1955, Bozeman, Mont.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. E. Koebbe
Secretary-Treasurer: Dr. Paul L. Peterson, 702 Sharp Building, Lincoln, Neb.
Time and Place: May, 1956, Lincoln

ACADEMY OF MEDICINE OF NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Francis J. Grant
Secretary: Dr. William A. Sturman, 219 South Harrison Street, East Orange, N. J.
Time and Place: 8:45 p.m., second Monday of the month, October to May. The Academy of Medicine, 91 Lincoln Park, South, Newark

NEW JERSEY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. James S. Shipman
Secretary: Dr. Albert F. Moriconi, 438 Hamilton Avenue, Trenton 9, N. J.

MEDICAL SOCIETY OF NEW JERSEY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Joseph R. Weintrob
Secretary: Dr. John Scillieri, 660 Broadway, Patterson 4, N. J.
Place: Atlantic City, N. J.

NEW JERSEY OPHTHALMOLOGICAL SOCIETY
President: Dr. George P. Meyer
Secretary: Dr. Charles E. Jaeckle, 136 Evergreen Place, East Orange, N. J.

NEW MEXICO OPHTHALMOLOGICAL SOCIETY
President: Dr. Howard B. Peck
Secretary: Dr. J. A. Dillahunt, 106 Girard S.E., Albuquerque, N. M.
Time: Meetings four times a year

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. George B. Ferguson
Secretary: Dr. James D. Stratton, 1012 Kings Drive, Charlotte, N. C.
Time and Place: September 12, 13, and 14, 1955, Columbia, S. C.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Merrill Reeh
Secretary: Dr. Ralph N. Westfall, 601 Medical Arts Building, Portland 5, Ore.
Time and Place: Fourth Tuesday ecah month, Henry Thiele Restaurant

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. James H. Delancy
Secretary: Dr. Daniel S. DeStio, 1006 Highland Building, Pittsburgh 6, Pa.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Jewell W. McLean
Secretary: Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C.
Time and Place: September 12, 13, and 14, 1955, Columbia, S. C.

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. O. E. Ballou
Secretary: Dr. Thomas Bryan, 2105 Hayes Street, Nashville, Tenn.
Time and Place: April, 1956, Memphis, Tenn.

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. E. Jackson
Secretary: Dr. Gatlin Mitchell, 1604 Medical Arts Building, Fort Worth, Texas
Time and Place: December 2 and 3, 1955, Galveston, Texas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Howard L. Mitchell
Secretary: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia
Time and Place: November 29 to December 2, 1955, Richmond, Va.

WASHINGTON STATE MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Walter C. Cameron
Secretary-Treasurer: Dr. Claude K. Miller, P.O. Box 1627, Wenatchee, Wash.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Ben W. Bird
Secretary-Treasurer: Dr. Frederick C. Reel, 404 Medical Arts Building, Charleston, W. Va.

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Otis M. Wilson
Secretary: Dr. G. L. McCormick, 6505 Central Avenue, Marshfield, Wis.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. F. Johnson
Secretary: Dr. John Scuderi, 145 W. Portage Trail, Cuyahoga Falls, Ohio
Time and Place: 6:30 p.m., first Monday in month. Akron City Club

ALTA CALIFORNIA OPHTHALMOLOGIC SOCIETY
President: Dr. Yale Brody
Secretary: Dr. Robert L. Alexander, 1127 Eleventh Street, Sacramento, Calif.
Time and Place: Monthly from September to May, Sacramento, Calif.

ALUMNI ASSOCIATION NEW YORK EYE AND EAR INFIRMARY
President: Dr. Webb Chamberlain
Secretary-Treasurer: Dr. Joseph Krug, 988 Fifth Avenue, New York, N. Y.
Time and Place: Annual Spring Meeting each year in April. New York

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Buford O'Neal
Secretary-Treasurer: Dr. Nathan I. Gershon, 727 W. Peachtree St. N.E., Atlanta 3, Ga.
Time and Place: Fourth Monday of each month except December, Academy of Medicine

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION
Chairman: Dr. Frederic M. Reese
Secretary: Dr. Herman K. Goldberg, 807 Cathedral Street, Baltimore 1, Md.
Time: Third Thursday of November, January and March

BALTIMORE NOSE AND THROAT SOCIETY
Chairman: Dr. Albert Steiner
Secretary: Dr. Walter E. Loch, 1039 N. Calvert Street, Baltimore, Md.

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY
President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, 501 Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through April. THE CLUB—Top of Red Mt., Birmingham

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC SOCIETY
President: Dr. B. Riseman
Secretary: Dr. Arthur Gorney, 14 Charlesgate West, Boston, Mass.
Time and Place: Annual meeting in March, Boston City Hospital

BROOKLYN OPHTHALMOLOGICAL SOCIETY
President: Dr. Donald E. Swift
Secretary: Dr. Nicholas P. Tantillo, 1 Hanson Place, Brooklyn 17, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB
President: Dr. Meyer H. Riwchun
Secretary: Dr. Elizabeth P. Olmsted, 568 Lafayette Avenue, Buffalo 22, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George Woodruff
Secretary: Dr. Clarence A. Fleischli, 415 S. 7th St., Springfield, Ill.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. James L. McGraw
Secretary: Dr. George A. Sisson, 713 E. Genesee St., Syracuse, N. Y.
Time and Place: Quarterly. University Club, Syracuse, N. Y.

CHATTANOOGA AND HAMILTON COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
Chairman and Secretary: Dr. C. H. Alper, 602-606 Georgia Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., third Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY
President: Dr. Norman Leshin
Secretary: Dr. Stanton A. Friedberg, 122 S. Michigan Avenue, Chicago 3, Ill.
Time and Place: First Monday of each month, Drake Hotel, Chicago, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY
President: Dr. Daniel Snydacker
Secretary-Treasurer: Dr. Frank W. Newell, 950 E. 59th St., Chicago 37, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB
Executive Committee: Dr. Donald J. Lyle, Dr. Charles K. W. Ascher, Dr. Barnet R. Sakler
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: First Wednesday night of month, November through May. Vernon Manor Hotel

CINCINNATI OTOLARYNGOLOGIC SOCIETY

President: Dr. Joseph Filger
Secretary: Dr. Edward T. Juler, 4140 Hamilton Avenue, Cincinnati 23, Ohio

CLEVELAND OPHTHALMOLOGICAL CLUB

President: Dr. Roscoe J. Kennedy
Secretary: Dr. Russell J. Nicholl, 10511 Carnegie Ave., Cleveland 6, Ohio
Time and Place: Four meetings a year, University Club, Cleveland, Ohio

CLEVELAND OTOLARYNGOLOGICAL CLUB

President: Dr. Irving I. Cramer
Secretary-Treasurer: Dr. William F. Hulse, 1314 Hanna Bldg., Cleveland 15, Ohio

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Vernon D. Stephens
Secretary: Dr. Trent W. Smith, 327 E. State Street, Columbus 15, Ohio.
Time and Place: First Monday of month, October through May, University Club.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. T. P. O'Brien
Secretary-Treasurer: Dr. Joseph A. Abbey, 429 Palmero, Corpus Christi, Texas
Time and Place: 7:00 p.m., first Wednesday of month, September through May, Nueces Hotel

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. F. Waldman
Secretary: Dr. M. Ray Harrington, 4317 Oak Lawn, Dallas, Texas

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Kirch
Secretary: Dr. R. R. Updegraff, 616 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:30 p.m., fourth Monday of each month, October through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL CLUB

Secretary: Dr. Frederick A. Lauppe, 1801 David Whitney Building, Detroit 26, Mich.
Time: Each month, November through April

DETROIT OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur P. Wilkinson
Secretary: Dr. J. Conrad Gemeroy, 664 Fisher Building, Detroit 2, Mich.
Time and Place: Fourth Thursday of each month, November through April, Harmoine Club, 267 E. Grand River, Detroit, Mich.

DETROIT OTOLARYNGOLOGICAL SOCIETY

President: Dr. Delmar Weaver
Secretary: Dr. Lyle Waggoner, 404 David Whitney Bldg., Detroit, Mich.
Time and Place: Third Wednesday of each month, David Whitney House

SECTION ON OPHTHALMOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

President: Dr. Robert L. Norment
Secretary: Dr. Robert E. du Prey, 1150 Connecticut Ave., N. W., Washington, D. C.
Time: Second Tuesday every other month

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

Chairman: Dr. Willard B. Walters
Secretary: Dr. Russell Page, 1835 I Street, N.W., Washington, D. C.
Time and Place: Third Tuesday, alternating months. Army and Navy Club

OPHTHALMOLOGICAL SOCIETY OF THE EAST BAY

President: Dr. Jay Sharpsteen
Secretary: Dr. Ernest E. Hessing, 483 30th Street, Oakland 9, Calif.
Time: Four times a year

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. John A. Cetner
Secretary: Dr. Frank C. Furlong, 713 Union Street, Schenectady, N. Y.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William Skokan
Secretary: Dr. W. P. Anthony, 708 Medical Arts Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month. St. Joseph's Hospital

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Ralph Kirsch
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Time and Place: Quarterly—February, May, October and December at Seven Seas Restaurant

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION

President: Dr. Claude C. Cody, III
Secretary: Dr. George M. Campbell, 1107 Hermann Professional Building, Houston 25, Texas
Time and Place: Second Thursday of each month, October through June. Houston Academy of Medicine Library Building

EYE, EAR, NOSE AND THROAT SOCIETY OF HUDSON COUNTY

President: Dr. Sidney Boyer
Secretary: Dr. Alfonse Cinotti, 3285 Hudson Boulevard, Jersey City, N. J.
Time and Place: Second Monday of each month, Jersey City Medical Center

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Russell A. Sage
Secretary: Dr. Lewis E. Morrison, 603 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of winter months, Indianapolis

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William B. Barry
Secretary: Dr. Dick H. Underwood, 1102 Grand Ave., Kansas City 2, Mo.
Time and Place: Third Thursday of the month, November through May. Usually at either Kansas City General Hospital or Kansas University Medical Center

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Sol Ludmerer
Secretary: Dr. George Zugsmith, 529 W. 8th Street, San Pedro, Calif.
Time and Place: Third Wednesday of every month September to May. Seaside Memorial Hospital

LOS ANGELES OPHTHALMOLOGICAL JOURNAL CLUB
President: Dr. Arthur S. Gray
Secretary-Treasurer: Dr. Jacob M. Kline, 3761 Stocker Street, Los Angeles 8, Calif.
Time and Place: Second Monday of each month. Los Angeles County Medical Building

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June. Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Robert A. Norene
Secretary-Treasurer: Dr. Leland R. House, 453 South Soto Street, Los Angeles 33, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May—Otolaryngology Section. 6:00 p.m. first Thursday of each month from September through May—Ophthalmology Section. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

LOUISVILLE EYE AND EAR SOCIETY
President: Dr. Joseph J. Wynn
Secretary: Dr. R. Glen Boles, 808 Heyburn Building, Louisville 2, Ky.
Time and Place: Dinner meetings at 6:30 p.m. on the second Thursdays of alternate months, 812 S. Second Street, Louisville, Ky.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time and Place: 8:00 p.m., second Tuesday in each month, September through June. Memphis Eye, Ear, Nose and Throat Hospital.

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. Erwin E. Grossman
Secretary: Dr. Howard V. Morter, 1714 E. Capitol Drive, Milwaukee, Wisconsin
Time and Place: 6:00 p.m., fourth Tuesday of each month, October through May, University Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Thomas Bryan
Secretary-Treasurer: Dr. Philip L. Lyle, 340 Doctor's Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. John M. McLean
Secretary: Dr. Joseph Laval, 136 E. 64th St., New York, N. Y.
Time and Place: 8:15 p.m., third Monday of the month, New York Academy of Medicine, 193rd St. and Fifth Ave., N. Y.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLARYNGOLOGY
Chairman: Dr. DeGraaf Woodman
Secretary: Dr. Otto C. Risch, 104 East 40th Street, New York 17, N. Y.
Time: Third Wednesday of each month

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Frederick H. Theodore
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York, 24, N. Y.
Time and Place: First Monday of the month, at 8:00 p.m., New York Academy of Medicine

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Clifford J. Blair
Secretary: Dr. Richard Wyrick, 812 Medical Arts Building, Oklahoma City, Oklahoma
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. James J. O'Neil
Secretary-Treasurer: Dr. Stanley M. Truhlsen, 1500 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Edmund B. Spaeth
Clerk: Dr. William E. Krewson, 3rd, 1930 Chestnut Street, Philadelphia 3, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, College of Physicians

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Wilfred E. Fry
Secretary: Dr. Thomas H. Cowan, 1930 Chestnut St., Philadelphia, Pa.
Time and Place: First Thursday of each month, October through April, Philadelphia County Medical Society Bldg.

PHILADELPHIA LARYNGOLOGICAL SOCIETY
President: Dr. William J. Hitschler
Secretary: Dr. Joseph P. Atkins, 3400 Spruce Street, Philadelphia, Pa. c/o U. of P. Hospital.
Time and Place: 8:30 p.m., first Tuesday of month, October through May, College of Physicians Building

PITTSBURGH ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OPHTHALMOLOGICAL SOCIETY
President: Dr. Fred Larimore
Secretary: Dr. Gertrude Baldwin, Greensburg, Pa.
Time and Place: 8 p.m., fourth Monday of month, Pittsburgh, Pa.

PITTSBURGH OTOLOGICAL SOCIETY
President: Dr. Louis L. Friedman
Secretary: Dr. John Dickinson, Mercy Hospital, Pittsburgh, Pa.
Time: Quarterly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. William H. Ludwig
Secretary: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month except June, July, August, December and January, usually in Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. W. Hancox
Secretary: Dr. James H. Parker, Jr., 238 N. 5th St., Reading, Pa.
Time and Place: Third Wednesday of each month, September through June (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Julius C. Hulcher
Secretary: Dr. Charles N. Romaine, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. J. C. O'Neill
Secretary: Dr. J. H. Leek, 205 W. Second St., Duluth, Minn.
Time and Place: Second Thursday of every month, at St. Mary's or St. Luke's Hospital, Duluth, Minn.

EAR, NOSE AND THROAT CLUB OF ST. LOUIS
Chairman: Dr. Robert E. Votaw
Secretary: Dr. Harry N. Glick, 1504 South Grand Boulevard, St. Louis 4, Mo.
Time: Third Wednesday in November, January, March and May at 6:30 p.m.

ST. LOUIS OPHTHALMOLOGICAL SOCIETY
President: Dr. James H. Bryan
Secretary: Dr. Philip T. Shahan, 508 North Grand Ave., St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Thursday of each month, October to April, except December, Park Plaza Hotel

SAN ANTONIO SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George B. Moore, Jr.
Secretary: Dr. Graham B. Milburn, 1617 Nix Professional Building, San Antonio, Texas
Time: Third Tuesday of each month, September through May

SAN DIEGO COUNTY OPHTHALMOLOGICAL SOCIETY
President: Dr. Wayne E. Monsees
Secretary: Dr. A. B. Root, 3315 Fourth Avenue, San Diego 3, Calif.
Time and Place: 6:30 p.m., University Club, second Monday of each month

SAN FRANCISCO COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Robert N. Shaffer, 490 Post Street, San Francisco, Calif.
Secretary: None
Time and Place: Last Tuesday of each month, February, April, September and November, San Francisco County Medical Society Building

SAN FRANCISCO OPHTHALMOLOGICAL ROUND TABLE
President: Dr. Robert N. Shaffer
Secretary: Dr. Albert Brinckerhoff, 490 Post Street, San Francisco, Calif.
Time and Place: San Francisco, three times a year

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Randall W. Henry
Secretary: Dr. Robert C. Maher, 512 Medical Center Building, Spokane 4, Wash.
Time and Place: 8:15 p.m., fourth Tuesday of each month, Paulsen Library Dentral Building

SUPERIOR CALIFORNIA EYE CLUB
President: Dr. Theodore Holstein
Secretary: Dr. John Berg, 436 Medico-Dental Bldg., Sacramento 14, Calif.
Time: Every third Thursday, eight months of the year

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. L. Reed Crammer
Secretary: Dr. Henry Drake, 316 Michigan Avenue, Toledo, Ohio
Time and Place: Second Tuesday of each month, Toledo Club

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. H. R. Mather, Jr.
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time and Place: Biannually. Third Thursdays of April and October. October meeting in Indiana, Pa.

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY
President: Dr. Charles J. Kistler
Secretary: Dr. Albert J. Abbott, 203 E. Green Street, Nanticoke, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street, Wilkes-Barre, Pa.

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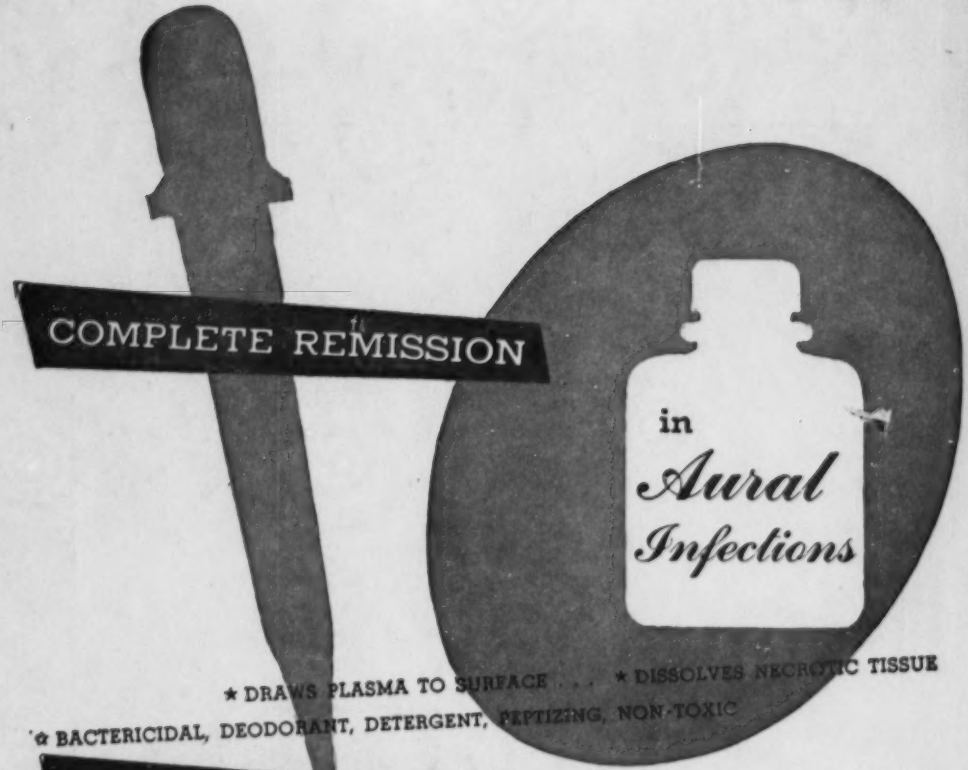

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